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GASTRIC ULCER ASSOCIATED WITH DIAPHRAGMATIC HERNIA*

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THE first description of gastric ulcer associated with diaphragmatic hernia was reported by Bright[1] in 1836. The patient, a young woman nineteen years old, died of exhaustion after a long period of vomiting and dyspnea. Autopsy revealed a congenital hernia of the diaphragm and ulcers on the lesser curvature of the stomach. Since this early date few cases of gastric ulcer associated with diaphragmatic hernia have been recorded. Extensive search has discovered the seventeen cases, listed chronologically in the table. In a few cases the patients reported typical symptoms of gastric ulcer; for the most part, however, ulcer was unsuspected and found only at operation or autopsy. Nowhere else does this lesion have such variety in its manifestations. One is impressed by the number of cases in which autopsy revealed an ulcer of considerable size which was overlooked at operation.

The following cases, reported in abstract form, furnish a background for comment on the genesis of gastric or duodenal ulcer in this region. According to Harrington[13] and Nord[16], they are superficial, in the nature of an erosion and do not penetrate the stomach wall. When the stomach has been replaced in the abdomen, the ulcers usually disappear and do not recur. According to Lindau and Wulff[17] the primary cause of typical gastric and duodenal ulcers would seem to be related to the biochemical conditions in the stomach. Hyperacidity of the stomach from too great secretion of acid gastric juice results in injury to the mucous membrane. There are histological zones of glands in the stomach and upper duodenum; the cardiac zone, the zone of the fundus glands, the pyloric zone, and the duodenal mucous membrane. The zone of the pyloric glands extends higher on the lesser curvature than on the greater. Ulcers appear more frequently in the area of the cardiac and pyloric glands where the gastric secretion is most active. If gastric juice has opportunity to act on the mucous membrane of the esophagus, ulcers may originate there. A predisposing factor is stagnation of the contents of an hour-glass stomach with regurgitation.

In many of the cases referred to above, the ulcers have perforated causing hemorrhage with secondary anemia. Microscopic examination revealed changes in the appearance of the gastric mucosa, most marked in those cases which presented a history of anemia or chronic bleeding. Kienbock and Möller[18] believe that chronic hemorrhage cannot be explained by simple incarceration of the stomach with resulting seepage of blood through the mucous membrane. Irritation of the stomach wall caused by constriction at the hernial orifice, however, might well cause hemorrhage, ulceration, and finally *carcinomatous degeneration*. Here we may find ulcer at the cardia degenerating into cancer so near the lower end of the esophagus that it would be recorded as carcinoma of the esophagus and not of the stomach unless the tissues were differentiated microscopically.

In many of the cases listed, the acidity of the stomach contents was determined by laboratory analysis. The usual site of the ulcer was on the lesser curvature in the zone of the cardiac glands. Sometimes, as in Hoffmann's[6] case, the site of ulceration included the pyloric zone. Knaggs[2] reported a case of true congenital diaphragmatic hernia with a 180 degree volvulus of the stomach and torsion of the omentum. Autopsy showed extensive hemorrhagic erosion on the internal surface of the fundus of the stomach. The stomach wall showed small multiple perforations and marked signs of strangulation. Evidently the stomach had passed freely in and out of the aperture in the diaphragm. In this attack, however, it became twisted with the omentum. The incarceration resulted in perforation and strangulation.

A similar case is reported by Dubs[3]. Moppert[4] describes in detail three cases of perforation of the stomach wall caused by incarceration of a portion of the stomach in the hernial orifice. Hernia of the diaphragm was not suspected as a possible cause of the lesion prior to operation.

Hamdi's[5] case was diagnosed as acute nephritis. Laparotomy revealed an inflated stomach of the hour-glass type in the right thorax. In the posterior wall of the fundus of the stom-

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CASES OF GASTRIC ULCER ASSOCIATED WITH DIAPHRAGMATIC HERNIA

No.	Author	Sex	Age	Side	Kind Hern.	Hiatus	Site of Ulcer	Kind Ulcer	Operation	End-result
I	Bright England 1836	F	19	R	Cong. true	Vena caval	Lesser curv.	3 chronic	No	Died 4 mos. after admission.
II	Knaggs England 1904	F	29	L	"	Esophageal	Fundus	Mult. perf.	Laparotomy	Died; 180° volvulus of stomach; strangulation.
III	Hamdi Germany 1905	M	50	R	"	"	"	7 chronic	"	Died at operation.
IV	Dubs Germany 1919	F	59	L	Cong. false	Paraesoph.	Lesser curv.		"	Died; volvulus of stomach, rupture.
V	Hoffmann Germany 1920	M	33	L	Traum. false	Esoph.	"	Large chronic	Thoracotomy Gastrojejunostomy	Died in hospital.
VI	Boysen Germany 1921	M	27	L	"	"	"	Large perf.	Laparotomy	Died in hospital.
VII	Plenk Germany 1922	M	44	R	Cong. true	"	"	Mult. perf.	Laparotomy	Died; volvulus, stomach; cong. short. esophagus.
VIII	Key Sweden 1924	M	30	L	"	"	"	Single chronic	Thoracotomy	Recovered.
IX	Schaper Germany 1925	M	30	L	Traum. false	"	Fundus	Single perf.	No	Died; erosion, branch of pulmonary artery.
X	Key Sweden 1926	F	63	L	Cong. false	"	Pylorus	Single chronic	No	X-ray diagnosis, no follow-up.
XI	Nord Sweden 1929	M	57	L	"	"	Lesser curv.	"	Neurectomy phrenic nerve	Recovered.
XII	Collier England 1929	M	55	L	Cong. true	Bochdalek	"	"	Thoracolaparotomy	Died in hospital; hour-glass stomach.
XIII	Collier England 1929	M	58	L	"	Esoph.	"	Single perf.	Gastrojejunostomy	Died in hospital; cong. short. esophagus.
XIV	Kronsjö Sweden 1929	M	60	L	"	"	"	"	No	Died in hospital.
XV	Harrington U. S. 1930	F	59	L	Cong. false	"	"	"	Gastrojejunostomy	Recovered.
XVI	Mathews U. S. 1931	F	68	L	"	"	"	"	No	Died in hospital.
XVII	Mathews U. S. 1931	F	62	L	"	"	"	"	Gastroenterostomy	Recovered.

ach were seven ulcers each the size of a penny. There was no pathological change in the pylorus or cardia.

In Hoffmann's[6] case, reported in 1920, a hernia in the diaphragm was ascribed to trauma. Laparotomy revealed a dilated deformed stomach. A gastrojejunostomy was done. Death occurred on the third day after operation. Autopsy revealed an ulcer the size of the "palm of the hand" occupying the whole of the lesser curvature from the cardia to the pylorus and spreading over both posterior and anterior portions of the stomach wall. Malignant disease was not mentioned in the report.

When the ulcer is small, it is easily overlooked especially if volvulus of the stomach is present. Boysen[7] reports that at operation he failed to find an ulcer the size of a nickel located on the posterior aspect of the lesser curvature about five centimeters from the pylorus. This patient had given a history of blood in the vomitus and stools for three years. At autopsy the perforating ulcer was found.

Plenk's[8] patient died of peritonitis from a perforating ulcer. A series of tiny perforations began seven centimeters from the pylorus and extended along the lesser curvature. The mucous membrane of the stomach was hypertrophied. The ulceration was interpreted as being caused by disturbed circulation from the volvulus of the stomach. Another complication was a congenitally shortened esophagus with atony.

Key[9] of Stockholm published the account of a case of gastric ulcer associated with diaphragmatic hernia in 1924. The patient, a man 30 years old, had suffered from chest discomfort for fifteen years. The condition was diagnosed by x-ray examination, which showed part of the stomach in the chest with a niche on the lesser curvature just above the diaphragm. Thoracotomy revealed the stomach firmly adherent to the lung, diaphragm, and pericardium. In freeing the adhesions a large hole was accidentally made in the stomach wall. This was sutured, the ulcer excised, and the crater cauterized. The stomach was then replaced in the abdomen and the hole in the diaphragm closed. The patient was well two years after the operation. X-ray examination at this time showed no ulcer present and the stomach in normal position. Key reports a second case of gastric ulcer at the pyloric end of the stomach associated with hernia of the diaphragm; the condition was discovered at x-ray examination and is not described further[10].

Collier[11] adds two cases to the literature on congenital diaphragmatic hernia associated with gastric ulcer. Both patients were male, 55 and 58 years old respectively. The former had suffered for ten years from tenderness and pain in the epigastrium. X-ray examination showed one-third of the stomach in the thorax. There was a large cavity in the stomach wall on the lesser curvature in the isthmus just below the diaphragm. Since the symptoms seemed due more to ulcer than to hernia, and operation seemed difficult and dangerous, medical treatment was begun. The patient was soon symptom-free. The x-ray plate showed no ulcer. Four months later, the patient returned to the hospital in worse condition than at the time of his first admission. He vomited dark material. X-ray examination revealed recurrence of the ulcer. By a combined thoracoperitoneal incision the stomach was found high and inaccessible and herniated through the left posterior part of the diaphragm. The mechanical difficulties encountered were almost insurmountable. The ulcer which measured $1\frac{3}{4} \times \frac{1}{2}$ inches showed no signs of malignancy. It was found impossible to close the aperture in the diaphragm completely. The patient died of shock twelve hours following operation.

In Collier's second case a man 58 years old was reported to have swallowed a penny when a child. This coin was retained in the esophagus and vomited up several months later (a suggestive manifestation of congenital diaphragmatic hernia in childhood). In 1922 he almost succumbed to an attack of severe hematemesis. In 1927 he suffered with indigestion, pain in the left chest and over the spleen. In 1928 occult blood was found in the stools. He was kept in bed on liquid diet with large doses of alkalies. At this time there was shoulder pain, hiccup, irregular pyrexia, and hematemesis. The hemoglobin fell to 42 per cent. The white blood count increased from 8,600 to 12,500. The diagnosis was made of chronic bleeding gastric ulcer densely adherent to the diaphragm. A jejunostomy was done and the patient improved as a result of jejunal feeding. X-ray examination later revealed a diaphragmatic hernia and large gastric ulcer. The radiograms were identical with those of the first case, except that in this case there was a congenitally shortened esophagus. The condition was thought to be inoperable and death occurred one month after the palliative procedure.

In the fall of 1929, Kronsjö reported the following case: A carpenter, 60 years old, was admitted to the hospital in the spring of 1927 with a history of vague gastric symptoms following an acute attack of bronchitis in 1925. In April, 1927, symptoms of vomiting, epigastric pain, dyspnea, and hematemesis became severe. His affliction was thought to be cancer of the stomach. X-ray examination with barium meal revealed over one-half of the stomach in the left pleural cavity with the left lung partially collapsed. There was a large opening in the diaphragm at the esophageal hiatus and marked posterior and lateral curvature of the spine. Operation was postponed.

Three months later the patient was read-

mitted. During this interval his condition had grown steadily worse. There was emaciation with marked signs of secondary anemia. There was a systolic murmur at the apex of the heart. The lower part of the chest was tympanitic, with dullness in the upper region. The urine was positive for acid and there was a marked acetone odor to the breath. The hemoglobin had fallen to 15 per cent.

The condition was diagnosed as perforating gastric ulcer associated with diaphragmatic hernia. It was apparent that the patient could not survive an operation. He was given a blood transfusion and kept on an ulcer diet. In spite of these measures he grew rapidly worse and died 25 days after admission to the hospital.

Autopsy revealed the stomach in a serous sac herniated through an esophageal opening which easily admitted three fingers. At the point of constriction on the lesser curvature a perforating ulcer as large as a florin was encountered. Erosion at the site had produced extensive pathological changes in the region of the ulcer crater.

Harrington[13] describes a case in his series of eight cases of diaphragmatic hernia associated with traumatic gastric erosion. His contribution is instructive in that it gives in detail, cases in all stages from superficial erosion of the gastric mucosa to definite formation of ulcer with well-differentiated crater. This case of gastric ulcer associated with diaphragmatic hernia occurred in a woman 59 years old. She was seen at the Mayo Clinic October 14, 1926. For twelve years she had complained of epigastric pain and dyspnea.

X-ray examination revealed herniation of the cardiac end of the stomach through the esophageal orifice and a small diverticulum on the lesser curvature. Laparotomy exposed a dilated, thick-walled stomach with numerous adhesions at the lesser curvature around a large gastric ulcer about 7.5 cm. from the cardia. Almost complete gastrectomy was done after the Pólya method of operation. Microscopic examination of the tissue excised from the stomach revealed a chronic, perforating gastric ulcer 3 cm. in diameter. The patient recovered.

In October, 1931, appeared two cases of gastric ulcer in the presence of diaphragmatic hernia, reported by Mathews and MacFee[14]. Both were in women, aged 62 and 68 respectively. The former, a nullipara, had been anemic for thirty years. In 1927 she suffered from severe epigastric distress and occasional attacks of vomiting. Both carcinoma and ulcer of the stomach were considered in the diagnosis. The hemoglobin ranged from 40 to 60 per cent. Operation done September, 1927, revealed a diaphragmatic hernia at the esophageal orifice through which a third of the stomach prolapsed. An ulcer crater, 2 cm. in diameter, was felt in the lesser curvature penetrating into the gastrohepatic omentum. When the hernial ring relaxed, the ulcer reëntered it and was just within the grasp of the ring of the diaphragm. Because of the position, resection was thought to be impossible. Gastroenterostomy was done. The patient recovered and reported four months after operation that she was free from symptoms.

The second case was recorded December 15, 1930. A woman, 68 years old, complained of constant dull pain in the right lower portion of the abdomen, weakness, and loss of appetite for four months. There was considerable distention of the stomach and constipation. Five years previously x-ray examination had revealed a diaphragmatic hernia with half the stomach in the thorax and much irregularity at the site of constriction. Gastric analysis showed relatively high acid contents of the fasting stomach. There was marked secondary anemia. The finding of blood in the stomach contents and in stools with the absence of any other lesion led to a diagnosis of gastric ulcer. On an ulcer diet the patient did well, but any change to semi-solid food caused a return of symptoms. At no time was operation thought advisable. February 20, 1931 the patient vomited dark blood and two days later occurred rapid collapse and death.

In these two cases a tongue of omentum crossed the herniated stomach and was attached to the ulcer base. The ulcer was found at the point where the lesser curvature was in contact with the constricting margin of the diaphragm. In other cases of diaphragmatic hernia observed by Mathews and MacFee, the congestion due to constriction of the diaphragm had caused mucosal bleeding without going on to the formation of actual ulcer. Thus it is logical to infer that the diaphragmatic hernia was the cause of the erosion and ulceration at the site.

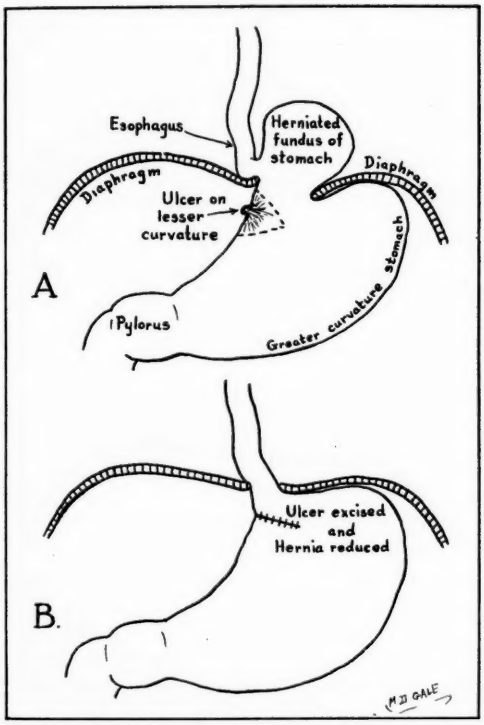
The following case is interesting because few symptoms of ulcer or diaphragmatic hernia were present.

Case No. 23,458. H. W., an unmarried woman, aged 52 years, was admitted to the Truesdale Hospital October 8, 1930 because of pain in the epigastrium. The patient stated that she had been in good health until 1920. At this time she had influenza followed by sinus infection. In 1922 she felt a gnawing pain in the epigastrium which occurred about twenty minutes after eating. She was also awakened at night by this epigastric distress, which was often relieved by sodium bicarbonate. These attacks seemed to have no seasonal periodicity but would last four or five months, cease for a time, and then recur. Of late they had become more constant and more severe. Recently the patient had dysphagia. There were no chills, fever, vomiting, nausea, or constipation, and no tarry or bloody stools. At times, however, stools had been clay-colored. The patient had maintained an average weight of 145 pounds.

At the time of admission the temperature, pulse, and respiration were normal. The systolic blood pressure was 138, the diastolic, 82. Erythrocytes numbered 4,500,000, leucocytes, 8,600 with hemoglobin estimated at 85 per cent. The Kahn test was negative.

Urinalysis showed an occasional pus cell and a faint trace of albumin. A roentgenogram of the gall bladder revealed a normally functioning organ. The stomach and duodenum series with barium meal and enema revealed an apparent dilatation of the lower end of the esophagus and slight delay in the passage of the barium meal through this region, findings consistent with cardiospasm. No other evidence of abnormality was found in the gastro-intestinal tract. The preliminary diagnosis was cholecystitis or the probability of gastric ulcer.

On October 11, 1930, the abdomen was opened by a right paramedian incision. Examination revealed an ulcer high on the lesser curvature of the stomach, and a paraesophageal hernia of the diaphragm. The ulcer itself was 2 cm. in diameter and located on the lesser curvature about 6 or 7 cm. from the cardia. The ulcer area was covered with omentum, increasing the area of induration to 5-6 cm. There



were enlarged glands in the gastrohepatic mesentery. The adhesions were freed and the ulcer excised by the Balfour operation. The stomach was closed with interrupted sutures of chromic catgut reinforced with continuous silk.

The opening in the diaphragm was triangular in shape admitting three fingers without difficulty. There was a hernial sac and no direct communication with the pleural cavity. The aperture in the diaphragm appeared inaccessible and no attempt was made to close it at this time.

The patient made a good recovery and on October 27, 1930, the aperture in the diaphragm was repaired by the thoracic route. The pleura was opened in the eighth interspace and the eighth rib reflected upward to afford adequate room. The seventh rib was also divided anteriorly at the midclavicular line. The phrenic nerve was exposed and injected with 1 c.c. of a two per cent solution of novocaine to gain temporary paralysis of the diaphragm. The hernial sac was found in the paraesophageal hiatus. It consisted of a thin membranous sac containing a portion of the stomach. The sac was obliterated by plication with a series of interrupted silk sutures, and the margins of the hiatus approximated.

X-ray fifteen months later revealed no sign of ulcer and the stomach in position below the diaphragm. The patient reported that she was free from symptoms.

SUMMARY OF CASES

A review of these 18 cases shows ten in males, eight in females. The left side was involved in 15. The ulcer was found 14 times on the lesser curvature of the stomach, usually about 7 cm. from the cardiac end. There were 5 inoperable cases; 4 patients died in the hospital. Laparotomy was done in 5, thoracotomy in 3, the combined operation in 1, gastrojejunostomy in 3, and neurectomy of the phrenic nerve in 1. Eight died after operation; five, 2 men and 3 women, recovered.

Hernia occurred through the esophageal hiatus 15 times. A sac was present in 9 cases. Three of the 9 false hernias were of traumatic origin, 1 from a fall, and 2 from gunshot wounds.

The type of ulcer varied from a small niche to multiple perforating ulcers. Hamdi[5], Dubs[3], Plenk[8], Knaggs[2], and Bright[1] report finding several ulcers on the lesser curvature or on the fundus of the stomach. In Schaper's[15] case, the ulcer eroded a branch of the pulmonary artery and caused fatal hemorrhage. In Plenk's[8] case, the ulcer perforated into the right pleural cavity. Death from hemorrhage caused by perforating ulcer occurred in Boysen's[7] and Kronsjö's[12] cases.

DIFFERENTIAL DIAGNOSIS

Disturbance of the gastro-intestinal function is revealed by the symptoms of epigastric pain, vomiting (often of the retention type), dysphagia, cardiac embarrassment, and, in cases of erosion of the stomach, hemorrhage. A fairly constant symptom accompanying cases of diaphragmatic hernia associated with gastric ulcer is secondary anemia. Therefore, it is probable that this condition will answer for one of the obscure causes of blood in the stools.

The symptoms are more often confused with those of cholecystitis than with any other lesion of the upper abdomen. In operations for cholelithiasis and cholecystitis whenever the gallbladder is found to be normal or nearly so, the diaphragm should be examined for hernia and the entire stomach for ulcer. Often the symptoms are not suggestive of ulcer. In the case which we report, there was no blood in vomitus or stools and no anemia.

In reviewing these cases, the following conclusions may be drawn. A strict ulcer diet does not lessen the severity of symptoms except for a very short interval. The patient can rarely take food an hour after the meal for relief of ulcer symptoms, because of the distress due to the diaphragmatic hernia. Treatment, then, is surgical. Since diaphragmatic hernia and gastric ulcer may coëxist and the former be the

forerunner of, if not a factor in causing the latter, it is wise to utilize every method of roentgenologic search for both of these abnormalities.

Symptoms caused by hour-glass constriction of the stomach ordinarily are not troublesome until there is interference with passage of food. However, when the stomach is so constricted that the upper chamber is above the diaphragm, in addition to symptoms of obstruction, there may be evidence of embarrassment of cardiac and respiratory function. When ulcer exists at or near the constricted area, it may escape detection by the ordinary methods of x-ray examination.

REFERENCES

1 Bright, R.: Account of a remarkable misplacement of the stomach. Guy's Hosp. Rep. 1: 598, 1836.

2 Knaggs, R. Lawford: On diaphragmatic hernia of the stomach and on torsion of the small omentum and volvulus of the stomach in association with it. Lancet. 2:358 (Aug. 6) 1904.

3 Dubs, J.: Hernia diaphragmatica vera paroesophagea mit volvulus und Ruptur des Magena. Deutsche Ztschr. f. Chir. 151:60 (Aug.) 1919.

4 Moppert, M. Gustave G.: La perforation de l'estomac par gangrène complication de la hernie diaphragmatique. J. de chir. 20:453, 1922.

5 Hamdi, Dr. med.: Der Magen als Inhalt einer rechtsseitigen Zwerchfellhernie mit sekundärer Ausstülpung nach der Bauchhöhle zu, eine rechtsseitige Pyonephrose vortäuschend. Deutsche Ztschr. f. Chir. 79:313 (Aug.) 1905.

6 Hoffmann, Viktor: Hernia diaphragmatica mit Ulcus ventriculi. München. med. Wchnschr. 67:986 (Aug. 20) 1920.

7 Boysen, I.: Beitrag zur Kenntnis des partiellen Magenvolvolus bei einem Zwerchfelldefekt, kompliziert durch ein blutendes Magengeschwür. Arch. f. klin. Chir. 117:768 (Sept.) 1921.

8 Plenk, Andreas: Zur Kasuistik der Zwerchfellhernie. Wien. klin. Wchnschr. 35: 339 (Apr.) 1922.

9 Key, Einar: Fall von Hernia diaphragmatica, kompliziert mit penetrierendem Magengeschwür. Zentralbl. f. Chir. 51:95 (Jan. 19) 1924.

10 Akerlund, Ake; Öhnell, Harald, and Key, Einar: Hernia Diaphragmatica Hiatus Oesophagei. Acta radiol. 6:1, 1926.

11 Collier, W.; Hurst, A. F., and Sheaf, E. W.: Two cases of gastric ulcer associated with congenital diaphragmatic hernia. Guy's Hosp. Rep. 79:159 (Apr.) 1929.

12 Kronsjö, M.: Diaphragmatic hernia of esophageal hiatus combined with gastric ulcer and fatal hemorrhage. Svenska Läk.-tidning 26:1067 (Sept. 13) 1929.

13 Harrington, Stuart W.: Diaphragmatic hernia associated with traumatic gastric erosion and ulcer. Surg. Gynec. Obst. 51:504 (Oct.) 1930.

14 Mathews, Frank S., and MacFee, William: Gastric ulcers dependent upon diaphragmatic hernia. Ann. Surg. 94:517 (Oct.) 1931.

15 Schaper, Kurt: Chronisches Magenulcus mit Arrosion eines Astes der Arteria pulmonalis bei transdiaphragmatischem Magenpiolaps nach Brustzwerchfellschuss. Deutsche Ztschr. f. Chir. 194:169, 1925-26.

16 Nord, Folke: Phrenic neurectomy as treatment of diaphragmatic hernia. Acta med. Scandinav. 72:511, 1929.

17 Lindau, Arvid, and Wulff, Helge: The peptic genesis of gastric and duodenal ulcer. Surg. Gynec. Obst. 53:621 (Nov.) 1931.

18 Möller, Folke: Diaphragmatic hernia and carcinoma of the stomach. Acta med. Scandinav. 74:421, 1931.

ACUTE PERFORATION OF PEPTIC ULCER*

Study of One Hundred and Six Cases

BY ANTHONY CORVESE, M.D.†

PEPTIC ulcer is one of the common and important medical and surgical problems and is said to occur in at least 10 to 12 per cent of the population. It is a serious condition not only because of the suffering and disability entailed but because of its grave complications, such as hemorrhage and perforation. This subject is of special interest to surgeons on hospital service where a goodly amount of emergency surgery is done. The incidence of perforation is given as from 4 to 20 per cent. Jacopo Penada of Padua, Italy, in 1793, was the first to describe the history and findings in a case of perforated ulcer of the duodenum. His patient, a man of 35 years, was suddenly seized with excruciating pain over the entire abdomen, followed by rigidity of the abdominal muscles. At autopsy a perforated ulcer on the first portion of the duodenum was found.

The following report is a study of 106 cases treated on the Surgical Service of the Rhode Island Hospital from January 1, 1925 to December 31, 1930. The operations were performed by twenty-one different operators and, because of the emergency, were frequently carried out by the junior surgeons late at night or in the early morning hours.

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Age and Sex: This group, as in other reports, shows the great preponderance of males, 96 (91%) over females, 10 (9%). The youngest was 19 years, the oldest 72 years. The actual age incidence is shown in Table I.

TABLE I

AGE INCIDENCE

Age	No. Cases	Age	No. Cases
19 to 24 years	10	49 to 54 years	11
24 to 29 "	17	54 to 59 "	8
29 to 34 "	15	59 to 64 "	0
34 to 39 "	17	64 to 69 "	4
39 to 44 "	9	69 to 74 "	1
44 to 49 "	10	Not recorded	4

Symptoms: The onset of perforation was sudden in all but two patients and the characteristic sign of severe abdominal pain with rigidity of the abdominal muscles and prostration was present in all but three cases.

Within the first few hours, although the patient looks very ill, the pulse and blood pressure are usually within normal limits. The patient is not in shock in the true sense of the word. Medical men must not be misled by the unaltered pulse rate and thereby cause serious delay. In the early hours the diagnosis is not difficult; in this group the correct diagnosis was made by the house officer in the majority

of cases. In a small number of cases there was a prodromal stage; patients had complained of stomach disturbance and the symptoms increased before the actual perforation. The history of acute onset while at work or following a heavy meal was fairly frequent. In two cases perforation took place immediately following x-ray examination with an opaque meal.

With the progress of the condition, the diagnosis becomes a little more difficult on account of the developing peritonitis. Here the pulse becomes rapid and signs of true shock appear. In making a differential diagnosis, the conditions most frequently considered are acute appendicitis, acute cholecystitis, acute pancreatitis, mesenteric embolus, tabetic crisis, volvulus and intestinal obstruction. The important factor is the recognition of the fact that the patient has an acute abdominal condition requiring immediate exploration.

Previous History of Gastric Disturbances: It is a well-known fact that in a certain number of cases perforation occurs without warning. In this study there was a history of gastric disturbance previous to the perforation in 88 per cent; in 4 per cent the symptoms had been present for from 1 to 2 weeks; in 14 per cent from 1 to 8 months; in 43 per cent from 1 to 17 years; in 23 per cent the duration of disturbance was recorded as indefinite time. In two cases there had been a previous perforation, one 3 years and the other 8 years previously.

Duration of Perforation: Minimizing the time which elapses between perforation and operation is one of the chief points in reducing mortality. Experience shows that early operations are safe and late operations are dangerous. The number of hours after perforation before operation was performed is shown in Table II.

TABLE II

RELATION OF DURATION OF RUPTURE TO MORTALITY

Hours Ruptured	No. Cases	Deaths	Percentage
2 to 4 hours	38	5	13
5 to 7 "	22	4	18
8 to 11 "	11	5	45
12 to 15 "	13	5	38
15 to 20 "	5	4	80
24 "	7	5	71
72 "	2	2	100
5 days	1	1	100
10 "	1	1	100
5 weeks	1	0	
No history recorded	5	1	20

Operative Procedures: The operation should be conducted as speedily as possible, preferably under spinal anesthesia. The perforation is located and closed with a mattress or purse-string suture of catgut. The question of the performance of gastro-enterostomy plus the closure has excited some controversy. It has warm advocates and warm opponents, Deaver among the former and Gibson the latter. From the literature I gather that gastro-enterostomy is being performed more often than formerly. However, the question of gastro-enterostomy depends upon the time elapsed since perforation, the condition of the patient, and the operator's skill. In this group simple closure of the perforation, reënforced with omental tag, was the method in 89 cases (85%). Drainage should not be used if avoidable. The actual operative procedures are given in Table IV.

TABLE III

RELATION OF LOCATION TO MORTALITY

	No. Cases	Deaths	Per Cent
Duodenal	72	18	25
Gastric	34	15	44

TABLE IV

OPERATIVE PROCEDURES

Type of Operation	No. Cases	Deaths	Percentage
Closure of perforation with drainage	80	23	28.6
Closure of perforation without drainage	9	2	22
Closure of perforation plus gastro-enterostomy without drainage	3	0	
Closure plus gastro-enterostomy with drainage	2	1	50
Closure plus appendectomy drainage	1	0	
Closure appendectomy no drainage	2	0	
Cauterization of ulcer, closure gastro-enterostomy	1	0	
Partial closure drainage	1	0	
Drainage of peritoneal cavity closure impossible	4	4	100
Drainage of subphrenic abscess	1	0	
Total cases drained	84	24	28.5
Total cases not drained	15	2	13.3

Location of Ulcers: Of the 106 cases, 72 (68%) were recorded as duodenal and 34 (32%) as gas-

TABLE V

CAUSES OF DEATH

Peritonitis	26
Secondary wound infection	1
Subphrenic abscess	1
Pulmonary T. B.	1
General sepsis, exhaustion	1
Lobar pneumonia	1
Pulmonary embolism	1

tric. Of the duodenal, 49 were on the first portion of the duodenum and 23 on the duodenal side of the pylorus; of the gastric, 19 on the stomach side of the pylorus, 12 on the lesser curvature, one on the greater curvature and two at the cardiac end.

As the figures show, the large majority (over 90%) of the perforations were on the anterior surface of the duodenum and stomach and within an inch or an inch and a half from the pyloric sphincter. Table III shows the relation of the location to the mortality and Figure I shows the location.

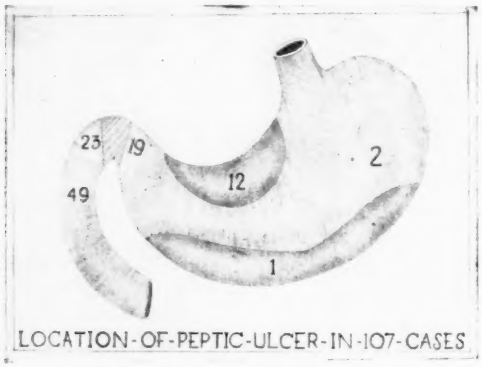

FIGURE I.
Should read 106 cases.

Postoperative Complications: The intra-abdominal complications were secondary incisional abscess, 1; rupture of the abdominal wound, 1; pelvic abscess, 2; subphrenic abscess, 1; secondary peritoneal abscess, 1; acute dilatation of the stomach, 1. (Table VII.)

TABLE VI

AUTOPSY FINDINGS

	No. Cases	Percentage
Peritonitis	8	100
Multiple ulcers	2	25
Cholelithiasis	2	25
Subphrenic abscess	1	12.5
Diaphragmatic hernia	1	12.5

Mortality: There were 32 deaths, 30 operative and 3 non-operative. In the gastric cases the percentage of deaths was almost twice as high as in the duodenal. Table III shows the relation of the location of the ulcer to the mortality. Table V shows the actual causes of death.

Autopsy Findings: Of the 32 deaths, in 8 autopsies were performed. The findings are shown in Table VI.

TABLE VII

POSTOPERATIVE COMPLICATIONS (ABDOMINAL)

Complication	No. Cases
Secondary abscess of incision	1
Rupture of abdominal wound	1
Pelvic abscess	2
Subphrenic abscess	1
Intra-abdominal abscess, secondary	1
Acute dilatation of stomach	1

SUMMARY AND CONCLUSIONS

(1) Of one hundred and six cases of acute perforation of peptic ulcer, over 90% were in males; 56% occurred between the ages of 20 and 40.

(2) Eighty-eight per cent gave a definite previous history of stomach disturbances. Of these very few had had x-ray examination.

(3) The large majority of the ulcers were located within an inch or an inch and a half from the pyloric sphincter; 72 were recorded as duodenal and 34 gastric. In the females there were more gastric than duodenal.

(4) In this series, simple closure of the perforation, with or without drainage, was performed with gratifying results in eighty cases. Whether more than this should be done depends on the time elapsed since perforation, the condition of the patient, the lesion present and the operator's skill.

(5) The operative mortality was 29%. This compares favorably with other reports. The mortality in cases of gastric perforation was almost double that in perforation of the duodenum.

(6) Acute perforated peptic ulcer demands prompt surgical treatment.

(7) When seen early, the diagnosis is comparatively simple.

(8) The responsibility of the physician who first sees the patient following perforation is grave, because the most important factor in successful treatment is early diagnosis followed by early operation.

(9) More frequent use of x-ray examination of the gastro-intestinal tract in patients with "indigestion" should be made.

EMERGENCY TREATMENT OF EXTREMITY FRACTURES

BY ROBERT H. KENNEDY, M.D.*

DURING recent years there has been an increasing demand for better treatment of fractures. There are several reasons for this. First, by the institution of follow-up clinics, it became evident that the late results in fractures were often indifferent or bad. The public had come to expect that a broken bone meant uniformly visible deformity, permanent disability and probably diminished earning power. In the second place, the adoption of federal and various state compensation laws brought home to industry the extent of loss which was going on through indifferent treatment.

Thirdly, increased efficiency in the economic management of industry showed that loss of time, permanent deformity and disability cut into the profits of industry. Improvement was imperative. Fourthly, the popularization of the x-ray by which the patient, the employer, juries, and Compensation Boards of Review could actually visualize the position of the fragments, made everyone feel that there must be improvement in treatment. In addition, the war gave a large number of surgeons an opportunity to treat fractures and become interested in them. Some of them brought back that interest and applied their methods to civil life. So we find that the public, the employer, the employee, administrative boards, and the medical profession are demanding improvement in fracture treatment.

Many general hospitals now have separate fracture services. In others the care of fractures is assigned to one of the most experienced rather than to one of the youngest members of the staff. In some hospitals visiting surgeons are called as soon as a patient with a fracture is admitted in the same way they would be for an acutely inflamed appendix. The immediate reduction of the fracture and the commencement of definitive treatment are important factors and have yielded satisfying results.

It is necessary, however, in at least one group of fractures to treat the injured man before removing him from the place where he was injured, namely, in fractures of the long bones of the extremities. Immediate traction is necessary to lessen pain and shock and prevent further damage. Overriding is characteristic of these fractures. As soon as the continuity of bone is broken, nature attempts to splint the part by muscular contraction. This allows progressive overriding, with possible further damage to muscle, vessel, or nerve from the fragments, or a simple fracture may be converted into a compound fracture. Under these conditions traction sufficient to neutralize the pull of the muscles is necessary. The commonly used long wooden side splint on a fracture of the femur, for instance, cannot exert traction and is so difficult to apply properly that even for immobilization it often does more harm than good.

During the later years of the World War the emergency treatment of fractures of the extremities was excellent, particularly in the American and British armies. No private in the Royal Army Medical Corps was allowed to go to the front unless he could apply a Thomas splint properly in a definite time. The American army improved the type of splint used and gave us the Keller-Blake hinged half ring splint. These were applied in the front lines under fire and no wounded man was allowed to be moved to the dressing station without one.

There seems to be no reason why civilian injuries of the same type should not be given as good treatment as was received during the war. The average citizen with training ought to be able to put on a splint as well as an army private.

With this in mind, several years ago we found at the Beekman Street Hospital, New York City, where we have a large fracture service, that while the visiting surgeons went at once to see patients with fractures, there were a good many patients who, we felt, had been further injured by not having some efficient splint applied before they were brought into the hospital. In 1926 we instituted traction treatment for all fractures of the lower and upper extremities before moving the patient from the place where he was injured. There is nothing new about this method. We simply insisted that as far as the ambulance service is concerned every fracture of the lower extremity from the hip joint through the lower end of the tibia and fibula, be put in a Thomas splint before the patient is removed. The same holds true for the upper extremity. Any patient with a fracture between the shoulder and the middle of the forearm is brought to the hospital in a traction splint. We now use the Keller-Blake hinged half ring splint for the lower and the Murray-Jones hinged splint for the upper extremity.

The splint is applied where the accident occurs. We frequently put it on at the bottom of an elevator shaft, on the deck of a ship, or on the street. In other words, we leave the patient where he is and apply a splint at once. The patient is not to be moved until sufficient traction is applied to prevent further damage. That is the point to be emphasized.

For the patient it increases comfort and lessens shock.

For the surgeon it simplifies reduction after

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the patient's admission to the hospital. We see few patients in whom there is any overriding of fragments as shown in the x-ray taken on arrival.

For the hospital as well as the patient it shortens his stay by promoting earlier union and there is less danger of non-union.

From the economic viewpoint the expense to industry is diminished because the injured man spends fewer days in the hospital and returns to work more quickly.

There are certain standards that are necessary in the application of this general method. It does not make any difference what particular procedure is used so long as one appreciates what it is sought to accomplish. Findlay[1] has described the method of traction which is used at Beekman Street Hospital.

In the use of traction we find that there are six requirements:

In the first place, there must be some adequate form of hitch, and it is necessary to protect the part beneath the hitch so that it will not be injured.

Secondly, the application of a traction hitch.

Thirdly, there must be some means of increasing traction by twisting so that the desired pull is obtained.

Fourthly, the extremity being in traction, it must be supported. One must not depend merely on traction for the entire support of the limb.

Fifthly, not only must it be supported from below, but lateral movement must be prevented.

Sixthly, the whole splint must be suspended in such manner that the heel will not, at any moment, rest on the ground, the floor of the ambulance, or elsewhere.

Suppose we are dealing with a possible fracture of the shaft of the femur, the standard application is as follows:

1. Cover the patient with blankets.
2. Administer morphine, if available, under medical supervision.
3. The foot, with or without shoe on, is grasped firmly by an assistant by both hands placed laterally.
4. Gentle, steady, firm traction is made in the axis of the extremity. Maintain manual traction until splint traction is established.
5. While traction is being made the whole limb is slowly lifted several inches from the ground.
6. Slip the half ring splint from the outer side beneath the extremity.
7. Make certain that the side bars are in the medial horizontal plane of the thigh and that the ring rests against the tuber ischii.
8. Buckle the strap over the anterior surface of the thigh.
9. Lower end of splint rests against the body of the assistant who is still applying traction.
10. Protect the ankle region. There are various methods. We use cotton wadding but a high shoe alone will suffice.
11. Apply traction hitch. We use ordinary cotton bandage with a square knot. The army traction strap or various hitches may be used.
12. Fasten traction hitch to the end of splint.
13. Increase traction by Spanish windlass action. We use two tongue depressors, but anything will suffice with which twisting can be done provided it is long enough to go over the side bars of the splint. At this point manual traction is released. The assistant has been maintaining traction up to this time so that the patient is relieved of pain.
14. Support extremity in splint. We accomplish this by muslin hammocks and clips applied before the splint is put on. Slings, bandages or anything else that will support the extremity after the splint is applied may be used.
15. Prevent lateral movement by muslin bandage above and below the knee or by carrying triangular sling used for support around extremity and splint.
16. Suspend end of splint to stretcher bar or to roof of ambulance—any method to keep the heel from resting on anything.
17. If the patient has to travel for twenty-four or thirty-six hours it will be better to have some sort of foot piece.

The time required for application of the splint to the arm is three minutes and is not over four minutes to the lower extremity.

Certain objections are urged against this treatment. First, that there may be no surgeon available. It is unnecessary that there should be a surgeon to apply it. When you realize the fact that the British and American privates did it, I do not think it is fair to say that anybody in first aid in industry, or a policeman or a fireman cannot be trained in its application just as well as a surgeon. The only thing is to appreciate the standards that are necessary.

A second objection is the expense. Splints are offered for from one dollar and a half to nine dollars. I would not recommend either the one dollar and a half one or the nine dollar one. I think the former probably is poor economy and the latter extravagance. There is a middle course between one dollar and a half and nine dollars where one may get a satisfactory splint. The Keller-Blake hinged half ring splint and the Murray-Jones hinged arm splint each cost the hospital six dollars and seventy-five cents, but they are both of better material than is necessary. The other supplies are no more expensive than one would have to meet in putting on a Liston splint or a Cabot splint. It is a matter of bandages and slings. At Beekman Street Hospital we use made-up washable muslin supports plus paper clips, because we find it less expensive to use the same articles several times.

Another objection is that it is unnecessary because the present methods are satisfactory. I

have seen many patients in Liston splints that were causing more harm to the patient than good. On the contrary we feel that traction is imperative. It is hard to say that present methods are satisfactory unless one agrees that it is proper to allow a fractured femur to await reduction until Nature has been trying in vain to initiate proper treatment for twenty-four hours or more.

Some say it is unnecessary for it is usually such a short haul to the hospital. That point is not well taken. If a patient is brought to our hospital by taxicab, and the nurse or interne thinks that there is a possibility of a fracture, immediately that patient has a Keller-Blake splint applied in the emergency room before anything else is done. The patient goes to the x-ray room or to the ward with this traction applied. The removal of his clothes is supervised so that if traction is interrupted at all it may be immediately reapplied. The distance does not count. As much damage can be done in the five minutes it requires to transport a patient from the x-ray room to the ward as at any other time. The main thing is to have the patient in traction as soon as possible.

Some say that it is dangerous in long hauls. We have used this traction method for eighteen hours in the hospital because the patient was not in condition to have anything done before that time. In those circumstances there is a ruling that the foot must be inspected every half hour, but it is rare that we have to release the traction even momentarily. It is not nearly so dangerous to have traction applied for eighteen hours as it would be to have the patient moved without proper immobilization of the fragments. Certainly, if a man is able to get sufficient training to put on the splints, there is no reason why he should not be able to inspect the foot and know whether the circulation is being impaired.

Then I am told it is dangerous in compound fractures. This does not hold, since the method was actually devised for compound fractures—it was developed in war times for use in bullet and shrapnel wounds. Our internes are trained, if they find a compound fracture, to use iodine on the wound and on projecting fragments of the bone. They make no attempt to reduce the fragment. If the fragment reduces itself on application of traction, that should occasion no worry. The patient must be operated on in any event and it seems no more dangerous to have the fragments in place with iodine applied than it is to have them projecting until treatment is started.

Traction treatment at the site of accident is being used in many places in connection with ambulances, police and fire departments. Some surgeons carry Thomas splints in their automobiles at all times. But with the widespread traffic accidents occurring at present, there is need for more general use of this treatment. It is a question of planning for equipment and instruction. Once tried the simplicity of its application and its obvious advantages make its use desirable.

SUMMARY

1. There is a demand for better treatment of fractures.
2. One neglected factor is traction treatment for extremity fractures at the site of accident.
3. The equipment and application are simple.
4. The manner of application may vary in certain details as long as accepted standards are followed.
5. These standards are outlined.
6. Various objections are answered.

REFERENCE

1 Findlay, Robert T. J.: Bone & Joint Surg. 13:701 (Oct.) 1931.

INTUSSUSCEPTION IN ADULTS*

BY GEORGE A. MOORE, M.D.†

THE mechanics of acute intussusception are practically identical in children and adults, but in few other characteristics does a similarity exist. A voluminous literature, in the past twenty years, has covered quite completely the incidenee and clinical manifestations of this disease in children. The scant attention which intussusception in adults has received in the literature gives one an erroneous impression of its incidence and gravity.

In 1909 Eliot and Corscaden[1] collected three hundred cases of intussusception in adults, giving a brief review of each case. Pitts[2] reported a total of one hundred and fifteen cases observed in St. Thomas Hospital, London, from 1875 to 1900; about 10% of these occurred in adults. McGregor[3] cited fifty-nine cases occurring in his practice prior to 1905, four of which were adults. Codman[4] collected twenty-seven cases from the records of the Massachusetts General Hospital for the ten-year period prior to 1908, nine of which were adults. In 1912 Kassemeyer[5] compiled two hundred and eighty-four cases of intussusception resulting from tumors, appendices and diverticuli. There were one hundred and fifty-one in his series over ten years of age and in eighty-nine the age was not recorded. Perrin and Lindsay[6] reviewed four hundred cases of intussusception from the records of the London Hospital, which were observed from 1903 to 1920, and found eighteen patients over fifteen years. Reisinger[7] collected thirty-eight cases of intussusception from the records of the Mayence Hospital, of which twenty-four occurred in

*Read at a meeting of the Plymouth District Medical Society, March 20, 1930.

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adults, and Starlinger[8] reported fifteen adults out of forty-six cases from the University of Vienna Hospital. Without more detailed information regarding the above statistics, it would appear to be a fair estimate that about five to ten per cent of all intussusceptions occur in adults. It is quite evident that no surgeon ever operates upon a large series of adults suffering from intussusception as is reported from surgical clinics in children's hospitals.

In a large proportion of children with intussusception no demonstrable cause of the disease is found, while in adults a tumor or some abnormality of the intestine is the cause in many cases. In Kassemeyer's series there were two hundred and twenty caused by tumors. Of the three hundred cases reported by Eliot and Corscaden about one hundred were due to tumors. In 1927 Clifton and Landry reported forty-five cases of fibromata of the small intestine. Intussusception was found in thirty-three of these cases at operation. Of fifteen cases of intussusception in adults cited by Starlinger, six were found to be caused by tumors.

Dividing the tumors causing intussusception into two large classes, Eliot and Corscaden found that of the one hundred cases which they had collected forty were malignant and sixty benign. Kassemeyer stated that out of one hundred and ninety-nine cases of intussusception resulting from tumors there were eighty-five of malignant origin and one hundred and fourteen benign. Of the malignant tumors in Kassemeyer's series, of the larger groups there were forty-eight carcinomas and twenty-six sarcomas and in the benign group sixty polypi and twenty lipomas. More recent writers who have recorded smaller series of cases are in accord with Kassemeyer's statistics. Theis[9] and Sherwood[10] have recorded cases of intussusception in adults caused by neoplasms of the ileocecal valve.

Meckel's diverticulum ranks next to tumors as a causative factor in adult intussusception. Eliot and Corscaden found thirty cases out of the three hundred which they compiled resulting from a Meckel's diverticulum. In Kassemeyer's series there were forty-two caused by Meckel's diverticuli. Hertzler and Gibson[11] in 1913 collected fifty-two cases of intussusception in adults resulting from Meckel's and in 1925 Lower added eight more cases from the same cause.

The vermiform appendix is a cause in a small percentage of adult intussusceptions and a smaller number result, from polyps, tuberculosis, intestinal ulcers and postoperative adhesions.

The comparative frequency of intussusception involving the small intestine alone and that involving the small and large as ileocecal and ileocolic, and those involving the large intestine only has been discussed by several writers. It is of some interest that Kassemeyer found ninety-two cases involving the small intestine alone as compared with one hundred and ninety-two cases in which the large intestine was involved in his series of cases. McIver[12] found thirteen cases of small intestine involvement in eighty-one intussusceptions, Perrin and Lindsay thirty-four intussusceptions of the small intestine in three hundred and thirty-five cases and Leichtenstern[13] one hundred and seventy-eight patients in whom the small intestine alone was involved in a series of five hundred and ninety-three cases. Of thirty-eight cases of intussusception reported by Reisinger, thirteen were located in the small intestine.

The classical picture of intussusception in infancy and childhood is rarely seen in adults except the occasional case in the acute stage of the disease. In most adult cases the symptoms extend over weeks, months and even years. Constipation of the most severe type is often observed in the chronic cases. Recurring attacks of colicky abdominal pains continuing for months occur in many cases. Vomiting is an uncommon early symptom. It occurs however in most of the late, neglected cases of complete obstruction. Blood in the stools in adult intussusception from any cause is of rare occurrence.

As a result of rigid infiltration of the intestinal wall in a certain number of adult intussusceptions due to malignant tumors, the lumen of the intestine remains patent until healthy intestine becomes involved.

Davis and Parker[14] state that x-ray studies of barium enemata are of great value in the diagnosis of certain cases of intussusception. A temporary obstruction to the column of barium is observed at the apex of the tumor, the barium later passing peripherally about the tumor, leaving a central gas-filled area. When complete obstruction has occurred, a V-shaped cupola of gas is observed above the column of barium.

Successful treatment of intussusception in infants and children, in most cases, is dependent upon early diagnosis, conservation of the child's strength and reduction of the intussusception. Resection carries an extremely high mortality in the early years.

The fact that a tumor frequently occurs as a causative factor of intussusception in adults adds to the gravity of the operation. To avoid recurrence in such cases, reduction must be supplemented by excision of the offending tumor as well as any other tumors of the intestine. Oughterson and Cheever[15] reported one case of intussusception in an adult and analyzed thirteen others in which from two to four operations upon each patient were necessitated on account of failure to excise the offending tumor or other tumors of the intestine.

The ideal method of removal of the tumor is by enterotomy and local excision. This method has been suggested by some writers and one case reported by Bevan[16]. Owing to the frequency of malignancy of tumors associated with intussusception, and the damage to the intestinal wall resulting in late cases, resection and anastomosis are necessitated in many cases.

The gravity of a resection in the treatment of acute intussusception in adults has not received the attention in the literature which it would appear to deserve. Reisinger reported twenty-four cases of adult intussusception, in nine of which reductions were accomplished with one death; resections were done in fifteen with six deaths. Starlinger cited thirteen resections in fifteen adult cases with a mortality of 33%. In Kassemeyer's series of two hundred and eighty-four cases there were seventy-three resections with death resulting in twenty-four cases.

The ideal treatment of intussusception resulting from a tumor, as shown by Oughterson and Cheever, is excision of the tumor at the first operation. It would appear, however, from the above statistics that resection and anastomosis should be reserved for the less critical of the acute cases. About the only recourse that we have at present to avoid resection when the intestine is damaged beyond viability is excision of the tumor or damaged intestine, leaving the two open ends of the intestine outside the abdomen. Most writers on intestinal obstruction in the past, however, have advised against this method on account of the high mortality which attended its use and the difficulty in eventually establishing continuity of the intestine. McIver and Shedden have used this method with successful results at the Massachusetts General Hospital.

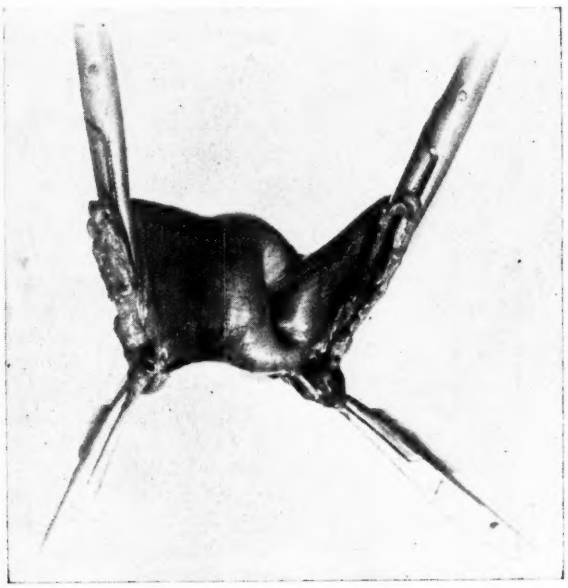
On account of the prolonged symptoms of intussusception frequently observed in the adult, many cases are operated upon before acute obstruction occurs. This fact, together with the ability of adults to withstand major surgery better than children, has been the main factor in lowering the mortality rate in adults.

CASE 1: G. S., a man aged 75, entered the hospital, September 20, 1929. For the past five years he had had gradually increasing difficulty in voiding, but had no attacks of retention. About six months ago he had an attack of colicky abdominal pain with marked obstipation and nausea without vomiting, lasting four days. Since then there had been occasional crampy pains in his abdomen and a laxative had been required frequently to obtain a satisfactory movement. No blood had been observed in the stools. He had lost no weight.

September 17, while on an auto trip through the White Mountains, colicky abdominal pains had recurred, gradually increasing in severity. He took a large dose of laxative, had a good movement the following day and was relieved. He then drove home, a distance of 200 miles. The day before entrance he felt quite comfortable but had no movement. That night the pain recurred accompanied by nausea. There was no abatement of the pain the next morning and he had vomited once.

Examination showed a rather thin, shaky old man curled up in bed on his side, complaining of severe abdominal pain. Abdomen level and moderately tender over the left lower quadrant. No mass was palpated. His rectum was normal except for a rather large, smooth prostate. Blood pressure 155-90. There was nothing else of importance in the examination.

Operation under spinal anesthesia: Right paramedian suprapubic incision. Coils of moderately distended small intestine presented in the wound. An intussusception of the ileum about four feet from the ileocecal valve was found which was six inches long. This was easily reduced and at its apex there was a tumor about the size of an English walnut.



CASE 1. Adenocarcinoma of Ileum showing mechanism of intussusception.

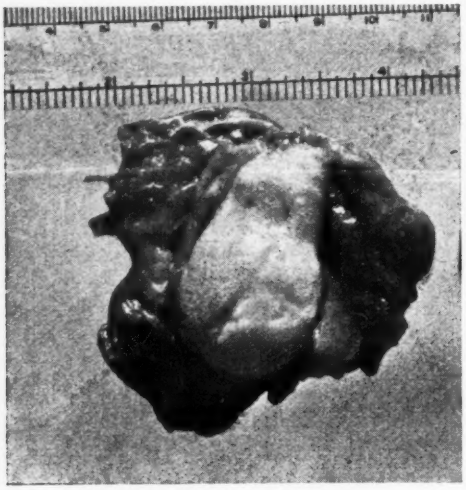
CASE 1. Adenocarcinoma of Ileum.

No glands were found in the mesentery. Four inches of the ileum was resected and an end-to-end anastomosis done. His convalescence was without incident.

Pathological report: Adenocarcinoma, primary, of ileum.

CASE 2: J. O., a woman aged 65, entered the hospital, November 30, 1930, with a history of having been constipated for the past eight years, requiring a laxative every night. She had had hypertension for several years and had been in bed for the four weeks previous for "heart trouble". Soon after breakfast, the morning before entry, she was seized with crampy, epigastric pain and became nauseated. At ten o'clock that night vomiting began and continued at intervals of one to two hours. The abdominal pain had not abated and the day of entry occurred in paroxysms.

On examination she had a foul breath, coated tongue, poor teeth. Heart sounds irregular, marked systolic murmur, lungs clear. Her abdomen was symmetrically distended, an oval, rounded, smooth mass about six inches long was felt in the left lower quadrant, which could be pushed over to the right of the median line with ease.



CASE 2. Lipoma of Ileum.

Operation under spinal anesthesia. Right paramedian suprapubic incision. Numerous coils of markedly distended small intestine presented in the wound. The tumor, an intussusception of the ileum was located. The constriction of the upper end of the intussusception was extremely tight so that reduction appeared to be quite difficult. The neck was gradually stretched with the finger coated with vaseline (a method which I have found to be of great value), after which reduction was accomplished with ease. On reduction, a soft tumor was found at the head of the intussusception, about 1½ by 1 inch in size, which was causing a dimpling of the wall of the intestine. Four inches of intestine was resected and an end-to-end anastomosis done. Two days after operation the patient developed double pneumonia and died the following day.

Pathological report: Lipoma of ileum.

Derocque[17] in 1924 collected one hundred and four cases of lipomas of the intestine and stated that there are two varieties, the submucous and subserous types. Bland-Sutton[18] described a polypus type which arises in the subserous layer, penetrates the muscular coat and causes a dimpling of the gut, similar to the case here reported.

Derocque found that 34% of the lipomas of the small intestine and 9% of those of the large intestine gave no symptoms; that in those causing symptoms, intussusception was found in 85% of the cases involving the small intestine and 50% in which the large intestine was involved. Comfort[19], in 1930, collected twenty-seven cases of lipomas of the small and large intestine from the records of the Mayo Clinic. Only three of these were producing symptoms requiring intervention; one was discovered at operation and twenty-four were found in 3924 autopsies. In a review of the literature Comfort found one hundred and fifty-three cases reported, making a total of one hundred and eighty-one cases.

SUMMARY

1. The incidence of adult intussusception is reviewed.
2. The frequency of tumors as a causative factor is shown. Meckel's diverticulum and other causes are mentioned.
3. Chronicity of symptoms in adults as compared with those in children is discussed.
4. Treatment of adult intussusception is reviewed.
5. Two adult cases are submitted.

REFERENCES

1 Eliot, E. and Corscaden, J. A.: Ann. Surg. 53:169, February, 1911.
2 Pitts: Cited by Eliot and Corscaden.
3 McGregor: Cited by Eliot and Corscaden.
4 Codman: Cited by Eliot and Corscaden.
5 Kassemeyer, E.: Deutsche Ztschr. f. Chir. 118:205-264, 1912.
6 Perrin, W. S. and Lindsay, E. C.: Brit. J. Surg. 9:46, July, 1921.
7 Reisinger, M.: Deutsche Ztschr. f. Chir. 227:255-271, 1930.
8 Starlinger, F.: Wien. klin. Wchnschr. 44: 143-145, Jan. 30, 1931.
9 Theis, F. V.: Ann. Surg. 87:676-683, May, 1928.
10 Sherwood, W. A.: S. Clin. North America 7:1057-1068, August, 1927.
11 Hertzler and Gibson: Cited by McIver.
12 McIver, M. A.: New Eng. J. Med. 199:453-456, Sept. 6, 1928.
13 Leichtenstern: Cited by McIver.
14 Davis, K. S. and Parker, C.: California & West. Med. 31: 330-335, November, 1929.
15 Oughterson, A. W. and Cheever, D.: Surg. Gynec. Obst. 48:682-686, May, 1929.
16 Bevan: Cited by Clifton and Landry.
17 Derocque, A. and Derocque, P.: J. de chir. 24:163-172, August, 1924.
18 Bland-Sutton: Cited by Derocque.
19 Comfort, M. W.: Proc. Staff Meet., Mayo Clin. 5:92, April 2, 1930.

A NONOPERATIVE TREATMENT OF CARBUNCLES*

BY WILLIAM A. WHITE, JR., M.D.,† AND EDWARD A. COONEY, M.D.†

MORE than five hundred carbuncles were treated at the Boston City Hospital during the four-year period ending November, 1931. Twenty per cent of these were treated in a manner which, though not new, is not widely known. This method is essentially one of permitting the lesion to run its course aided by nonspecific foreign protein therapy; and so striking and gratifying have been the results that we have come to regard it as the treatment of choice.

In a clinical lecture in 1869, Sir James Paget, after experience with more than two hundred cases, made a plea for the nonoperative treatment of carbuncles, based on the following conclusions: first, incision does not prevent extension; secondly, it fails to allay pain in most

*Read at a Clinical Meeting at the Boston City Hospital, February 18, 1932, by the Department of Surgical Research and the Harvard Surgical Teaching Service.

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cases; and thirdly, the process of healing is not hastened.

We believe that conservative treatment of carbuncle of the upper lip is now a generally accepted surgical principle. Incision in these lesions is contraindicated because of the danger of spreading the infection to the brain. Yet, in this group of cases, the disease runs its course and the patients uniformly do well. The traditionally high mortality attributed to this location has not been evidenced in our series.

Various other forms of conservative treatment have had their adherents. Pierre Delbet has employed heterovaccination since 1913, since which time, Peterson says, no case of carbuncle on Delbet's service has been treated by surgical incision. Others have preferred specific and autogenous vaccines; still others circuminjection of autogenous blood; and more recently x-ray therapy has been highly recommended. It is possible that the success of these measures is due largely to protein therapy plus restraint of the surgeon's hand. Indeed, according to Holthusen, small doses of Roentgen rays cause massive destruction of the lymphocytes, and the split bodies thus originated account for some of the effects of x-ray therapy. Poultices, carbolization, Bier's vacuum hyperemia, ionization, high frequency, ultraviolet ray, and other accessory measures have had their enthusiastic supporters, all of which would seem to indicate that carbuncle runs a natural course, and probably would get well with little or no treatment.

What is this natural course? The pathology of these lesions is too well known to be discussed in detail. For our purposes, however, we regard carbuncle as a kind of cellulitis which eventuates in central necrosis and slough. Cellulitis in its various forms is not usually benefited by operation, because frank pus, as from an abscess, is rarely obtained. The infection enters through the pilo-sebaceous apparatus of the skin, and is usually evidenced by a small pustule soon to be surrounded by a painful, red induration which may or may not progress extensively. Thus we have the first stage: subcutaneous cellulitis, demarcation of the edges, and a heaping up from edema. After a few days the second stage follows with freedom from pain, central necrosis, liquefaction, and discharge of slough and pus. The third stage is that of healing with granulation and epithelialization after cessation of infection.

By protein therapy we believe we fulfill the requirements recently set down by Light and Sosman. They said: "If a treatment can be found which will shorten the cycle by eliminating or hastening the onset of any . . . stages, or one which will relieve the pain . . ., or one which will effect a healing without surgical interference, saving the patient both operation and later scar, this will be deemed an improvement in the known methods of treatment."

First founded on purely empirical observations, nonspecific protein therapy was introduced about twenty years ago. The procedure was greatly simplified in 1916 when Schmidt and Saxl recommended milk injections. This was followed by a wave of popularity which reached its peak eight or more years ago. Many investigators have sought a scientific explanation of this phenomenon but the resulting theories are numerous and complicated.

Müller expressed the opinion that the fundamental action of parenteral protein injection lies in stimulating the immunizing activity of the bone-marrow. This is evidenced, he said, by the development of fresh, granular, neutrophilic leucocytes, and an increase of specific protective bodies in the serum, the effects of which are apparent at the site of injection. There is said to be an increased activity of all the cells of the body, with resultant changes in blood and lymph vessels, nerves and local tissues. Excitation of the sympathetic nervous system causes vasodilation and greater permeability of the capillaries. The detoxicating influence of the liver is augmented; and the lymph flow, as measured in the thoracic duct has been found to be increased four-fold. The diluted toxins are digested by enzymes. Autolysis and suppuration are accelerated by this extension of the body's inherent powers of resistance.

This action is manifested clinically by a pronounced euphoria,—a feeling of well-being. In from two to eight hours, in most cases, pain is markedly diminished or ceases entirely; appetite is increased; sleeplessness is banished, and there is a very definite diminution of toxic symptoms. Coincident with this there is a moderate leucocytosis, and in some cases an elevation of one or two degrees in temperature. We have never observed an untoward reaction at the site of injection, nor were there symptoms of protein shock in any of our cases.

One is greatly impressed by the rapid local response to the treatment. All stages in the cycle are affected. There is early demarcation of the lesion and abrupt limitation of extension. Intense reddening and tumefaction are followed in a few days by softening, central necrosis, and sinus formation. The liquefaction is astonishing, the carbuncle seeming literally to melt away. The liquefied slough is discharged through surprisingly small, cribriform openings, or the openings coalesce, allowing the core to be lifted out *en masse*. Incision is unnecessary. Even granulation and epithelialization appear to be speeded up owing to general cell excitation.

It has been our privilege to analyze more than five hundred cases of carbuncle treated in this clinic, and to separate from them 110 in which protein therapy was employed at some stage of the disease. For purposes of comparison, we found it necessary to classify the lesions according to relative size. This we have done by terming small lesions No. 1, large No. 4; 2

and 3 are medium small and medium large respectively. In only 208 cases were we able to determine accurately the duration of disease from onset until complete healing had occurred. These cases are divided into three groups: those treated by surgical incision, those by incision plus protein, and those by foreign protein without incision. The accompanying table shows the duration in days of disease in the three groups.

TABLE I

AVERAGE DAYS OF ILLNESS IN VARIOUS SIZED CARBUNCLES IN 208 CASES TREATED WITH AND WITHOUT NONSPECIFIC PROTEINS

Size of Lesion	No. 1	No. 2	No. 3	No. 4
Surgery	12	29	54	74
Surgery plus Protein Therapy	—	26	39	69
Nonspecific Protein	15	21	26	45

It will be noticed that the protein cases, the second and third groups, ran generally shorter courses, with the exception of the No. 1, or very small carbuncle. In defense of our technique in this wayward group, however, we would point out that in 280, or all lesions incised or excised, 14 per cent suffered extension, and required one or more further operations. With nonspecific therapy, extension is checked in the early stages; a No. 1 carbuncle remains a No. 1. In 4 per cent of the surgical cases, moreover, skin graft to complete cure was advised, as compared with none in our series.

Because of their convenience, sterility and uniformity, we have used commercial ampoules of nonspecific protein exclusively. These agents were Aolan, "a germ and toxin free lactalbumin solution, made from cow's milk"; Omnadin, "a combination of proteins, lipoids and fats," and Activin, "a purefied 5 per cent casein in combination with organic iodine". At present our preference is for the first named, aolan, although we have had satisfactory results with them all. Inasmuch as we have used it from the beginning and in more cases than the others, we are more familiar with its application. Its simplicity appeals to us. Dosages vary, as with any therapeutic agent, according to the response elicited; over-dosage seems impossible; the course of the disease and the sensations of the patient are the best guides. Treatments are intramuscular into the gluteal or deltoid regions; and, in our series, type 4 carbuncles have averaged but four injections.

Patients are often mystified because the active treatment seems to be directed to an area remote from the seat of trouble. Partly for this reason, and partly because it may help, we treat the lesion locally. In one case, however, a dry sterile dressing was all that was used. We favor intermittent applications of moist heat as long as inflammation is present. Hot chlorinate dressings, hot glucose packs, or enzymol, are valuable when the lesion is open. In many cases, at this stage, we have employed a water-soluble jelly containing bacteriophage, but we have not been impressed by its use, nor do we believe that x-ray should be combined with this treatment. It is difficult to evaluate the worth of various antiseptics, for none stand out. In one case we experimented with maggots to remove the slough. The maggots grew fat, did their job expeditiously, but caused considerable pain. Balsam of Peru, five per cent silver nitrate solution, shingle strapping with adhesive, and other methods are sometimes beneficial in the third or healing stage. Then, too, general supportive and eliminative measures are to be recommended. Most of the patients need not be hospitalized, or if so, for a few days at the most.

We believe that our treatment has certain distinct advantages over what we choose to call the old method. From the standpoint of the patient the treatment is ideal. He need no longer fear operation or anesthesia,—considerations of no small importance to many, and rightfully so since the aged, the debilitated, and the cardiac withstand operation poorly. Then, too, there are economic factors; this is an ambulatory treatment; constitutional symptoms are quickly and surely removed, permitting the patient to resume his normal activities much earlier than if he were operated on. A minimal scar, often invisible, is obtained. From the physician's standpoint, very little skill, as compared to that in operative procedures, is required. The ease and safety of administration recommend this technique.

A word might be added concerning carbuncle complicated by diabetes. Many surgeons have already agreed with us up to this point. Some argue that the pernicious interrelation between infection and diabetes demands immediate surgical removal of the infection. We yield to their point of view, providing it is possible to remove the infection, and in removing it, not endanger the life of the patient. We would point out, however, that so-called surgical drainage of a carbuncle usually results in very little drainage; also, that the diabetic, even with insulin, is still a poor surgical risk. Moreover, large carbuncles in this group of patients have been treated by this nonoperative method, and with considerable success.

SUMMARY

To summarize, we have studied more than 500 cases of carbuncle, twenty per cent of which had parenteral injections of nonspecific protein. Paget's results in 200 cases, and the course of lip lesions are offered as evidence that carbuncle follows a natural cycle. Besides, we have come to believe that surgical interference prolongs the infection. Since this is so, a method which will increase the body's defensive mechanism against

the disease is logically indicated. Protein injection favorably activates cell stimulation and increases the specific protective bodies, thus aiding nature. We believe that this is the treatment of choice.

CONCLUSIONS

1. Carbuncles run a natural course.
2. Nonspecific protein therapy assists this course, making operation unnecessary.
3. Spread of infection is checked, pain is relieved within two to eight hours, and healing is hastened.
4. The technique is simple.

ILLUSTRATIVE CASES

CASE I. Hospital number 659107.

A Polish barber, aged 60, was treated on the Fifth Surgical Service by courtesy of Dr. Irving J. Walker. A carbuncle on the back of the neck started December 24, 1931, as a pea-sized swelling. The patient was admitted to the hospital on January 4, 1932 with a size No. 3 lesion, triangular in shape,

CASE I. Photograph No. 1 shows a fourteen-day-old No. 3 carbuncle, after the third daily injection of Aolan. Demarcation, tumefaction, and beginning central necrosis are present.

CASE I. Photograph No. 2 was taken at the end of the fifth week. The clean crater and narrow, granulating wound are evident.

measuring nine by five by six and one-half centimeters. There was considerable induration, and two small pustules. Daily injections of Aolan (10 c.c. intragluteally) were given with marked diminution of pain eight hours after the first treatment. There was no local treatment except a dry gauze dressing applied to the back of the neck. Two weeks after the onset of the illness, or after the third injection (see Photograph No. 1), definite demarcation, tumefaction, and central necrosis appeared. For experimental purposes, this patient was given fifteen injections without any untoward reaction. Daily leucocyte counts were done before and two hours after injection and showed a constant increase of from two to four thousand cells. On the seventh day after admission, the lesion had completely liquefied, was draining freely, and the patient's temperature remained normal. On the sixteenth day, again experimentally, a cage containing 300 maggots was applied to expedite the removal of slough. This

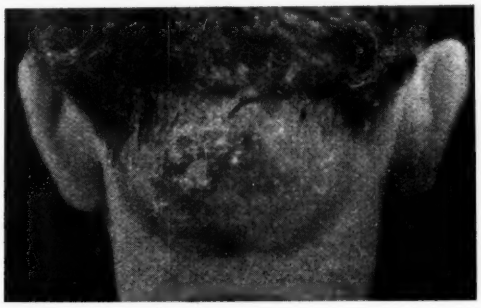
CASE I. Photograph No. 3 demonstrates the early end-result with minimal scarring, seven weeks from the start of the disease.

treatment made the patient very uncomfortable and is not to be recommended in the routine case. Photograph No. 2, taken on the day of the discharge from the hospital, one week after the maggots were applied, shows a clean shallow crater. The wound was dressed twice in the Out-Patient Department, and was entirely healed February 2, 1932, forty days from the date of onset. Photograph No. 3 was taken two weeks later and shows an almost invisible scar.

In commenting on this case, we wish to point out that this patient was purposely overtreated and without deleterious effects. Probably four daily injections, with a fifth four or five days later, would have been sufficient.

CASE II. Hospital number 660112.

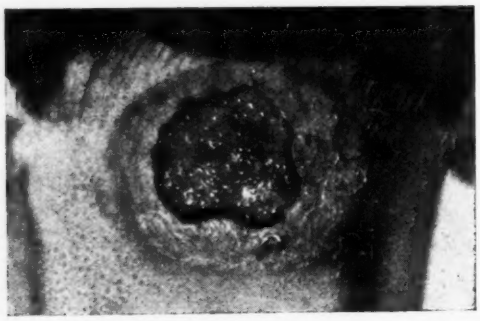
A sailor, aged 47, was admitted to the Fifth Surgical Service on January 14, 1932, seven days after the onset of the disease. He presented a No. 4 type of carbuncle covering almost the entire back of his neck. On admission, his temperature was 102°F.;



CASE II. Photograph No. 1 was taken the day after admission, and shows a well-defined, indurated No. 4 lesion, eight days after the onset.

his leucocyte count was 18,700, and his urine was not abnormal. He said that he had had no sleep for five nights. Two hours after the injection of ten

cubic centimeters of Aolan intragluteally, his pain ceased. Photograph No. 1, taken the day after admission to the hospital, shows a large indurated lesion with definite demarcation of the edges. The patient was given daily injections of Aolan. On the sixth day his temperature was normal and the carbuncle was draining freely. The lesion was treated locally by applying hot flaxseed poultices



CASE II. Photograph No. 2 shows a clean, shallow crater two weeks after treatment with Aolan.

CASE II. Photograph No. 3 illustrates the end-result two months after the onset of the disease.

superimposed on sterile gauze saturated with hot, dilute, chlorinated soda solution. Photograph No. 2, taken twenty-two days from onset, shows a clean shallow crater. After that time, the patient was treated in the Out-Patient Department until discharge. Normal epithelialization took place, and the wound was entirely healed on March 9, 1932, sixty-two days from onset. (See Photograph No. 3.) Commenting on this case, we would like to point out that this patient was given more injections than were necessary and made a much longer stay in the hospital than is usual. Time will iron out the wrinkles in his scar making it practically invisible.

CASE III.

A janitor, aged 61, was admitted to the Second Surgical Service on February 8, 1932, and was treated by us, through the courtesy of Dr. R. C. Cochrane. We classified his carbuncle as a type No. 3, the induration being three inches in diameter on the left side of the back of the neck. The onset dated from February 3, 1932—one week after he had recovered from "grippe". On admission, his temperature was 100°F., leucocyte count was 14,000, and his urine not abnormal. He said that he had been unable to sleep for three nights. Shortly after admission, four cubic centimeters of "Activin" were injected into his left deltoid muscle, with cessation of pain (in the region of the carbuncle) in eight hours. Softening and liquefaction set in on the following day. The lesion was treated locally with hot chlorinate dressings and flaxseed poultices. Three more injections of two cubic centimeters of "Activin" were given on successive days. The liquefied slough and pus discharged through multiple cribriform openings, leaving an absolutely clean crater, nine days after treatment was started. The carbuncle was entirely healed with almost no scar on March 9, 1932, thirty-four days from onset.

CASE IV. Outpatient No. 236208.

A male cook, aged 55, came to the Outpatient Department for treatment for the first time on October 9, 1931. He said that nine days before, a furuncle had appeared in his right temporal region. When first seen by us, he showed a dome-shaped mass, dry and indurated, about two inches in diameter. Four cubic centimeters of "Omnadin" were injected into his left deltoid muscle and the patient was told to apply hot flaxseed poultices to the dressing over the carbuncle. On the next day, the patient reported that his pain stopped six hours after injection; Omnadin injection was repeated and again three days later. On the fifth day after treatment was begun, the carbuncle was soft and beginning to liquefy. Daily dressings of Staphylojel ointment and sterile gauze were applied. The slough was lifted out *in toto*, October 21, 1931, and on November 2, 1931, thirty-three days from onset, the patient was discharged entirely healed with a very small scar.

REFERENCES

Paget, James: Clinical lecture on the treatment of carbuncle. Lancet 1: 73-75 (Jan. 16) 1869.

Peterson, William F.: Protein Therapy and Nonspecific Resistance. New York, The Macmillan Co., 1922.

Carp, Louis: Treatment of carbuncles. Ann. Surg. 86: 702-706 (Nov.) 1927.

Holthusen, H.: Biologische Wirkung der Röntgenstrahlen mit Berücksichtigung therapeutischer Fragestillungen. Klin. Wchnschr. 1[1]: 766 (Apr. 15) 1922.

Anderson, A. J.: Large carbuncle of neck cured by high frequency currents. Am. J. Dermat. and Genito-Urinary Dis. 13: 218, 1909.

McIlhenny, Paul: Bier's vacuum hyperemia in the treatment of carbuncle. New Orleans M. & S. J. 59: 29-33, 1906-07.

Dodds, W. T. S.: Autogenous vaccine in carbuncle of the lip and face. M. Council 15: 10, 1910.

Frick, William: Etiology and treatment of carbuncle. J. Missouri M. A. 7: 88, 1910-11.

Martin, Walton: Surgical Infections. Dean Lewis' Practice of Surgery. (Hagerstown, Md., W. F. Prior Company, Inc.) 1: (Chap. 10) 49-52, 1928.

Delafield, Francis, and Prudden, T. Mitchell: Text-Book of Pathology. Ed. 11. New York, William Wood & Co., 1919.

Light, R. U., and Sosman, M. C.: The treatment of carbuncles by roentgen-ray. New Eng. J. Med. 203: 549-555 (Sept. 18) 1930.

Lewis, R. W.: A conservative treatment of carbuncles. Ann. Surg. 78: 648 (pt. 2) Nov. 1923.

Tenenbaum, J. L.: Protein therapy, with special reference to skin diseases and syphilis. Therap. Gaz. 39: 311 (May 15) 1923.

Müller, E. F.: Berl. klin. Wchnschr. 56[2]: 801, 1919. München med. Wchnschr. 67: 9, 1920. 68: 912, 1921. 69: 1506, 1922. 69: 1753, 1922. 71: 202, 1924.

Miller, H. E., and Epstein, N. N.: Non-specific protein therapy in dermatology. California & West. Med. 27: 500-503 (Oct.) 1927.

Ahlswede, E.: Nonspecific protein therapy. Arch. Dermat. & Syph. 5: 586-588 (May) 1922.

TRICHOMONAS VAGINALIS VAGINITIS*

BY JOE VINCENT MEIGS, M.D.†

INTRODUCTION

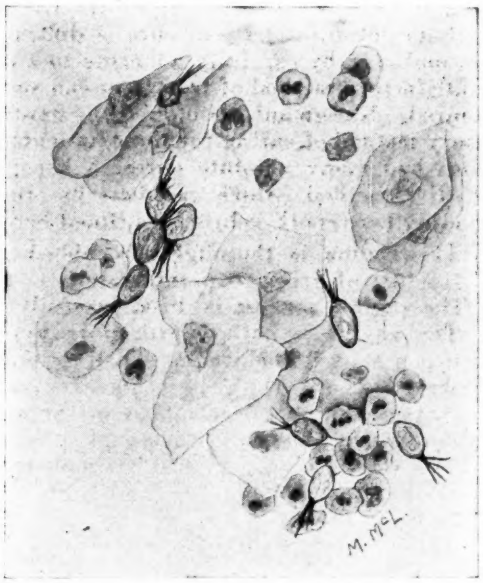
VAGINAL discharge, or leukorrhea, is a very common complaint. The cause of the discharge depends upon the etiological factor. In many cases the correct curative procedure is definite; for instance, cauterization or amputation of the cervix clears up chronic endocervicitis and repair of a lacerated cervix very often stops a disagreeable discharge. However, the secretion due to vaginal infection with the trichomonas vaginalis is very difficult to check. This flagellate, for many years recognized as a cause of vaginal discharge, is often seen in the secretion of patients without marked symptoms. In a recent study Bland et al.[1] found trichomonades in 22.7% of pregnant women and only 13.2% complained of an irritating discharge. It is even found occasionally in the urine. It is, however, undoubtedly the etiologic factor in many cases of profuse and very disagreeable leukorrhea. Eradication of the organism from the vagina causes a complete cessation of symptoms. Thus proof of its pathogenicity is offered. The source of the organism may be the intestinal tract, although Bland believes that it is not.

SYMPTOMS

The symptoms caused by the growth of this organism in the vagina are very typical. The history given by the patients often suggests the diagnosis. A profuse and sometimes a moderate discharge that causes itching and burning, with a very characteristic "disagreeable feeling" in the vagina, is almost always due to trichomonades. There is often an odor to the discharge, but this odor is not characteristic. But few patients complain of frequency and burning of urination. The chief symptoms are a profuse discharge plus a "disagreeable feeling" in the vagina. Nearly every patient speaks of this "feeling" and a mention of it nearly clinches the diagnosis.

EXAMINATION AND DIAGNOSIS

On examination of the vagina one is at once struck by the foamy white or creamy discharge. The foamy appearance is due to the presence of many air bubbles. The vulva is often red and irritated. The vagina is the seat of a marked vaginitis and the cervix often shows small punctate areas of redness and ulceration. The foamy discharge is also found in the upper vagina surrounding the cervix. The cervical canal is not involved. This picture is not seen in other conditions. Verification of the diagnosis of trichomoniasis is quite simple. A moderate amount of the discharge is placed on a glass slide, a drop of warm water is added and a cover slip is placed over the mixture. The slide should be viewed almost at once under the high dry power of the microscope. No stain is used. The light of the microscope is cut down about one half to bring out the cells and organisms more effectively. Pus cells and epithelial cells are easily seen and often a great many small oval organisms are noticed, moving about through the pus and epithelium. The organisms are oval or round and at one end a few fast moving whips or flagella can be made out. Careful inspection of one



Drawing showing pus cells, epithelial cells and trichomonas vaginalis. The flagellae and outline of the organisms are overemphasized. The trichomonas is about the size of a pus cell and is easily confused with it. However, the small "whips" of the flagellate and its motion make differentiation easy.

of these organisms will at once make it clear that, although it is the same size and often the same shape as a pus cell, it is something entirely different. When there are but a few organisms present, if a clump of pus or epithelium is watched, a small amount of motion may be seen, and suddenly a trichomonas will be noticed trying to move about. By gently tapping the slide the trichomonas will be freed and easily identified. The diagnosis is very simple after a little practice with moist smears. Sometimes the whole microscopic field is filled by a mass of slightly moving organisms with their thin, small flagella moving very rapidly. The slides must be looked at immediately after the preparation is made for death comes quickly, and without characteristic motion it is hard to differentiate them and pus cells. Phagocytes full of motile bacteria are frequently confusing, as are pus cells

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with actively moving granules in the cytoplasm. These will be recognized easily after a little practice. It is very difficult to find the organism in the moist smears after the first treatment has been carried out. A stained smear is of no aid in making the diagnosis for the organisms do not take a stain satisfactorily. The moist smear is of utmost importance and from this type of smear the diagnosis is easily made.

TREATMENT

Patients with trichomonas vaginitis are often operated upon, cauterization of the cervix or amputation being the most frequent procedures. The results of surgical treatment are nil and the discharge returns soon after the patient leaves the hospital. Douches keep down the discharge as long as they are used, but upon cessation of treatment it reappears. Vaginal suppositories and various vaginal tamponades are helpful but do not cure. A real cure is difficult to obtain and only by carefully adhering to a tried and satisfactory method of treatment can success be assured. Kleegman[2] has offered the most satisfactory method of caring for these patients and she has had many absolute cures in her practice. Her method, which can best be studied by reading her report, is briefly outlined here.

1. The vagina is thoroughly scrubbed with tincture of green soap and water.
2. The vaginal mucosa is dried carefully.
3. The vagina is bathed with pure pyroligneous acid. The vagina must be put on stretch and the speculum frequently turned around to make sure all areas are reached by the acid.
4. The vagina is packed full of small lamb's wool tampons which are well coated with Lassar's paste.

In the acute stage she treats the patient three times a week and uses 2% mercurochrome instead of the green soap scrub and pyroligneous acid, but does use the lamb's wool tampons covered with Lassar's paste. When the mucosa is healed, two treatments a week are given and continued until the vaginal mucosa resembles skin. This takes place after six or eight weeks of treatment. Her patients are not considered cured until free of organisms for at least four months without any douching. Kleegman further recommends careful washing of the vulva and anal regions with green soap and water twice a day and the application of a calamine lotion to the vulva. The lamb's wool tampons are always left in place by the patient until the next treatment. She carries out her treatment through the menstrual period for at this time the organisms grow rapidly.

After having attempted to treat a number of cases by this method and not effecting any permanent cures, I consulted Dr. Kleegman and quotations from her letter show how carefully the treatment must be carried out. She believes that the therapy must be followed to the absolute letter to obtain results. She writes[3] that "the question of success or failure in this particular condition depends entirely on attention to detail. Do you turn the speculum all round so as to reach every portion of the vaginal mucosa? Do you have the lower half of the table well elevated so as to have the solution reach and saturate the vault of the vagina? Do you treat your patients right through the menstrual period? This is most important—daily during these days if possible. Are your patients scrupulously conscientious about their external treatment twice a day and about thorough cleansing after a bowel evacuation? Are your tampons small enough so that they can be packed right into the fornices and do you use a sufficient number to thoroughly distend the entire vagina? Are they well coated with Lassar's paste? Our tampons are made of lamb's wool, quite small; the paste is buttered on while softened by moderate warming. How long do you treat your patients? How often? Do the tampons remain in place until they return to you for the next treatment? I do not use tincture of green soap and pyroligneous acid till all the ulcerations and erosions are healed. Till then in the acute stage, I use 2% mercurochrome followed by 2% silver nitrate—then the Lassar's paste packing". In her letter she states that she has had nearly 200 cases in private practice and has cured 87% of them. The treatment, then, is slow and painstaking, but good results can be obtained if her method is followed in all its details. It is unquestionably true that patients can be kept comfortable and organism-free as long as douches are kept up.

DISCUSSION

This report deals with but eight cases, but the miraculous symptomatic cure in these patients following immediately after the first treatment is its excuse. Fifty per cent of patients are free of symptoms and very grateful. The treatment used is a modification of Kleegman's. The expense entailed by such frequent office calls as her method demands is too great, and some means had to be found to eliminate this objection; also the relief experienced by patients is so marked that it is difficult to impress upon them the necessity of frequent visits. The modified treatment used is as follows: The vagina is thoroughly washed with the tincture of green soap and water and carefully dried. It is then painted everywhere with full strength pure pyroligneous acid and the vagina filled with soft tampons covered with Lassar's paste. The patient is given a prescription for a solution of oxycyanide of mercury 1-100 and is advised to use three teaspoonfuls of the solution to a quart of warm water as a douche morning and evening[4]. This dilution makes a 1-7000 solution. She is warned of the importance of thorough washing and cleansing of the vulva and anus with the tincture of green soap and

water twice a day and after a bowel movement. The office treatment is carried out twice a week for the first two weeks, then once a week for two weeks, and then once in ten days for two treatments. The patient is then instructed to use the douche once a day and report for reëxamination at the end of a month. If organism-free, the douche is omitted and a smear is observed at the end of another month. All patients were symptomatically cured but only half were free of the organism. One patient reported again after six months; she did not have any of her old symptoms but wished to report for reëxamination. She had taken an occasional douche of boric acid. This patient, although much better was not cured as numerous trichomonades were found in a vaginal smear. It is evident that this method of treatment, although satisfactory so far as the patient is concerned, is not so likely to effect an absolute cure as is Kleegman's method and her method is, therefore, recommended where it is economically possible to carry it out.

CONCLUSIONS

1. Trichomonas vaginalis vaginitis is a frequent cause of irritating leukorrhea.

2. This condition, usually suggested by the symptoms, can be easily verified by means of a microscopic examination of a moist smear of the discharge.

3. Kleegman's treatment, outlined above, is a method that will cure the infection.

4. Another method of treatment is described that has resulted in 50% of permanent cures and 100% of symptomatic cures.

5. The relief experienced by these patients is great and almost immediate.

6. Cauterization and amputation of the cervix for leukorrhea should not be recommended until trichomonas vaginalis vaginitis is ruled out.

REFERENCES

1 Bland, P. B.: Wenrich, D. H., and Goldstein, L.: Trichomonas vaginitis in pregnancy. Surg. Gynec. Obst. 53:759-767 (Dec.) 1931.

2 Kleegman, S. J.: Trichomonas vaginalis vaginitis, common cause of leucorrhea. Surg. Gynec. Obst. 51:552-555 (Oct.) 1930.

3 Kleegman, S. J.: Personal communication.

4 Dannreuther, W. T.: Some useful office procedures in gynecological therapy. New Eng. J. Med. 203:351-356 (Aug. 21) 1930.

IS THE "APPALLING INCREASE" IN HEART DISEASE REAL?

In the July issue of the *Journal of Preventive Medicine*. Bolduan publishes a paper under the above title based on a careful analysis of New York City's mortality statistics since 1900. The following summary and conclusions may be of interest to our readers.

SUMMARY

A study of New York City's mortality statistics has been made to determine the truth of the statements that there has been an "appalling increase in the death rate of heart disease."

The character of the basic data (death certificates) is analyzed and sources of errors to be guarded against are pointed out.

The difficulty of classifying deaths in which there is more than one cause of death is discussed, and the practice of statistical offices is described.

The pathological changes and the etiological factors in heart disease are shown not to be limited to the heart, but to include also the arteries and kidneys.

The registered increase in heart disease is admitted and shown to be accompanied in New York City by a registered decrease in apoplexy, Bright's disease and deaths charged to senility.

Statistics are presented which show that in New York City, for the population as a whole, the death rate from cardio-arterio-renal disease is no higher now than it was thirty years ago.

Similar statistics are presented for the four important age groups, and only in the group aged 65 years and over do these show any increase in the death rate from cardio-arterio-renal disease.

CONCLUSIONS

1. In New York City, for the population as a whole, there is no evidence of any increase in the real death rate from heart disease.

2. The registered rise in the death rate from heart disease is largely, if not wholly, fictitious.

3. Statistics based on the registered deaths from heart disease alone are fallacious. Deaths registered under apoplexy, arterial disease, kidney disease and senility must be taken into account.

4. The great prevalence of cardio-arterio-renal disease presents a very important health problem. Its solution demands accurate statistics.

5. In the United States registration area as well as in New York City, the specific death rates even in the higher age group have declined since the beginning of the century, a fact which it is difficult to reconcile with any considerable increase in the mortality of heart disease.

6. A detailed statistical study of the course of the death rate from cardio-arterio-renal disease in the United States registration area should be made, due consideration being given in such a study to the various sources of error discussed in this paper.

Until such an analysis demonstrates that for the registration area there has been an actual, significant increase in the death rate from cardio-arterio-renal disease in the various age groups, we shall continue to regard the "appalling increase" in heart disease as largely a myth.

THE HEALTH DEPARTMENT'S ATTITUDE

There appears to be some misapprehension concerning the Health Department's attitude regarding statistics from heart disease. The fear is expressed that to group deaths from heart disease with cardio-arterio-renal disease represents a step backward, and creates a statistical scrap heap from which no information can be obtained. This fear is based on a misconception of the purpose of the grouping. We are strongly of the opinion that only the group figures should be considered when one is attempting to determine whether or not "heart disease" is on the increase, but we do not for a moment maintain that statistics should not be tabulated for all the various components of the cardio-arterio-renal group. On the contrary, we feel every effort should be made to present these in as detailed a manner and as accurately as possible.—*Bulletin N. Y. City Department of Health*, August 13, 1932.

THE NEW HAMPSHIRE MEDICAL SOCIETY

APPENDICITIS*

BY JAMES B. WOODMAN, M.D.,† FRANKLIN

IF the death-rate in appendicitis were improving even by a fractional percentage it might not seem necessary to again harp on this much hackneyed subject. Such, however, is not the case. There has been no improvement in our mortality figures for appendicitis during the past twenty years and in the past five years we have not quite held our own. In fact, the death-rate is slightly higher than it was five years ago.

The Government death-rate for appendicitis in 1927 was 15 per 100,000. In 1928 it was 15.2. In New Hampshire in 1927 there were 51 deaths from this cause. The rate gradually rose until in 1930 there were 74 deaths or 15.9 per 100,000. In the *Journal of the American Medical Association* for February 12, 1932, the following summary was given; these reports were from 150 hospitals and comprised the work of more than 1,500 surgeons with over 83,000 cases and the final results may be summarized as follows: General mortality, 4.909%; acute cases, 4.88%; chronic cases, .99%; suppurative cases, 10.15%; gangrenous cases, 8.77%; cases with spreading peritonitis, 21.19%.

In view of the fact that very many, perhaps a majority of these deaths seem avoidable if operated earlier, I believe that no apology is necessary for going briefly over some of the salient points of this situation with the purpose of crystallizing certain facts which are well-known but sometimes neglected.

Appendicitis is not only the commonest surgical condition, but contributes more to the general mortality than any other purely surgical condition. In persons over one year of age it is the commonest abdominal emergency.

It is a disease especially of childhood, youth, and early adult life, for which reason this high death-rate is all the more regrettable. Its mortality is largely preventable. While the quality of surgical treatment plays its part, the most essential factor in lessening the death-rate from this cause is prompt and accurate diagnosis.

To be sure, in some cases of appendicitis the infection may be so mild and the circulation so good that the process may subside spontaneously without extending beyond the appendix itself and it is probable that in some few cases no further attacks follow. I believe, however, that the scar tissue caused by such an attack produces a narrowing of the lumen of the appendix and that subsequent attacks are more than likely to occur, and these generally appear with increasing severity. The possible adhesions incident to any attack may also impede the circulation thus predisposing to further trouble. Doubtless there are cases in which the increased tension in the appendix finally forces the infected contents into the cecum and spontaneous recovery takes place in this way. Very commonly there will be an extension of the infective process through the appendix wall to the surrounding tissue and a more or less local inflammatory process will be set up which will bring about the formation of a mass of omental tissue with the appendix in its centre. Even this will occasionally absorb in the course of from four or five days to a week. More often, however, this condition will be followed by the formation of a walled-off area with the formation of an abscess and the presence of a broken down appendix in the wall of the abscess. This is the condition which we find about the third or fourth day in the cases with active inflammation. Of course there is no certainty that the patient is going to be fortunate enough to have a walled-off abscess and then we have a spreading peritonitis. Some cases seem to have no tendency whatever to wall off and thin pus will be seen invading all parts of the abdominal cavity. These are the cases which show the highest mortality and the patient too often dies as the result of a general peritonitis. More than one out of every five of this type will die.

In the cases where a well walled abscess has formed, it may be wiser simply to drain the abscess and remove the appendix at a later time if it is not readily accessible, as the dissecting out of an appendix which is a part of the wall may break open the well-sealed defense and allow infection to spread through the abdomen. I remember an appendix that was removed from a case in which an abscess had been opened and drained at three different attacks covering a period of three or four years.

When an attack of appendicitis is in progress any one of the above-mentioned results may follow. No diagnostician will be able to prophesy which it will be.

With these facts in mind I have made four general rules for the management of appendicitis which it seems to me may be worth while:

First. Appendicitis is a surgical disease and its treatment is surgical. While this may not be universally admitted and there may at times and in cases be some contraindicating condition, the acceptance of this statement as a fixed rule will, in my opinion, save many lives.

Secondly. After a reasonably sure diagnosis can be made, the sooner the appendix is removed the better the patient's chance of recovery. This

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should cut down the number of spreading peritonitis cases with its 21% mortality.

Thirdly. No case in which appendicitis may be the diagnosis should be given castor oil, salts, or laxatives of any sort. Also the gastro-intestinal tract should be given complete rest. Food to some extent, and laxatives to a greater extent, set up peristalsis and it is a noteworthy fact that a large percentage of the ruptured appendices which come in to us have been given strong laxatives.

Fourthly. In a case of suspected appendicitis morphine should not be given as it masks the symptoms and gives the patient and the doctor alike, a false sense of security which will be the means of losing valuable time while the process may be developing to a dangerous stage.

It is well to bear in mind that the condition of the appendix may be found much worse than the symptoms would lead one to expect and many times have we rejoiced over taking out a severely infected appendix in the middle of the night rather than waiting until the morning. No harm can be done by early operation provided, as in any surgical procedure, that the diagnosis is correct.

Diagnosis. In the typical case a few clear cut points in history and physical examination should tell the story.

History. Acute appendicitis ordinarily occurs during otherwise normal health, without prodromal symptoms, although it may occasionally be preceded by some feeling of malaise or vague abdominal discomfort and perhaps some disturbance of the bowels. The *first* symptom is *pain*, which is usually of moderate severity but of a rather sickening quality. This pain may be practically anywhere in the abdomen but will commonly be referred at first diffusely to the region of the umbilicus or epigastrium. Or it may start in the right iliac fossa and remain there throughout. This pain may be relatively mild and is generally not of the intense character of renal colic. It may, however, be intermittent. In cases where the appendix is distended and under great pressure, the pain may become quite intense. Very often within a short time after the onset of the pain there will be nausea and, frequently, vomiting of food or bile. This does not relieve the pain as would be the case in some gastric disturbance. Generally within a few hours the pain shifts from the epigastric region and seems to center in the appendix region, while the diffuse abdominal pain decreases. This shift of pain is very characteristic of appendicitis.

The temperature and the pulse rate usually increase moderately by this time and the pulse may take on a snappy beat which is not normally present. The temperature is usually not over 102 or 103 and if it rises much above this point the diagnosis of appendicitis should be questioned. We have seen a number of persons with appendicitis with the appendix gangrenous who walked into the office with a temperature and pulse practically normal. In these cases, however, there was much pain and very decided tenderness and well-marked muscle spasm.

Along with the rise of temperature there may come a moderate leucocytosis. This is not absolutely diagnostic as sometimes it will not appear and it may also be caused by some other factor. It is, however, a confirmatory bit of evidence.

Often the bowels will be constipated in an attack, but if they move there is no accompanying relief from the pain as there should be were the pain caused by an accumulation of gas or by constipation. Now and then a case will have some looseness of the bowels but this is not so common.

The question of previous attacks of a similar nature should be determined, their number, dates, duration and severity. As already mentioned, these attacks, if recurrent, generally increase in severity.

It may be helpful in taking the history to ask the patient what he was doing at the beginning of the attack and thus focus his mind on the onset and learn the first symptom. Did the attack awaken him from sleep or cause him to quit work? What did he do next, and so on, thus getting the course of the symptoms as nearly as possible in their chronological order, as this is most important.

So much for the history.

Physical Examination. On inspection it may be noted that the right leg is drawn up so as to relieve the tension on the abdominal muscles though this will not always be present. The breathing may be thoracic rather than abdominal as the abdomen may be more or less splinted by muscle spasm. If there is intestinal obstruction, peristalsis and distention may be noted. Palpation gives the most valuable information of all. The important point to determine in the early stages of the disease is whether local abdominal tenderness and true muscular spasm are present. Palpation of the abdomen should be begun at a point well away from the suspected site of trouble, say in the upper left hand quadrant. It should be gentle and slow. The two sides of the abdomen should be compared and a gradual approach should be made to the lower abdomen. Hyperesthesia of the skin should not be mistaken for spasm. Hyperesthesia will give you a sudden muscular contraction on contact. Voluntary muscle contraction may be ruled out by getting the patient to relax by deep breathing with the mouth open. The entire abdomen should be carefully palpated including the flanks and the ileo-costal regions as gravel, ureteral kinks, floating kidney and pyelitis should not be forgotten. There should be local tenderness over the appendix and increased muscle resistance at this point.

In this connection it should be noted that while the proximal end of the appendix is fairly constant in its location, being anchored to the cecum, the distal end may be found anywhere from the liver border to the pelvis or in the flank to the outer side of the cecum. So while the average point of maximum tenderness will be half way between the anterior superior spine and the umbilicus there will be some variation from this for the reasons stated. While making palpation, if the patient's attention is directed in another direction it will often be possible to work down deeper in the right lower quadrant and it should be found that tenderness increases on deep pressure if the appendix is at fault, whereas that would not be the case if you were dealing with a pneumonia of childhood accompanied by a diaphragmatic pleurisy and consequent splinting of the abdominal muscles. Muscle spasm should be studied carefully. It is a constant, involuntary resistance, tightness or hardness of a muscle and should be determined by comparison with the corresponding muscle on the opposite side and in different parts of the abdomen.

Percussion is not conclusive. It may demonstrate gas forming over an abscess, but you will get about the same note from gas in the cecum. Auscultation is also inconclusive, although it may give evidence of increased peristalsis in cases of intestinal obstruction.

The order, then, of the appearance of the symptoms is of great importance. The sequence is *pain* first, then nausea, then rise in temperature and leucocytosis. Do not, however, insist on waiting for a rise in temperature as has already been pointed out. Sometimes it will not appear until brought on by an advancing peritonitis. If fever or nausea were present before the onset of pain, be suspicious that it is not appendicitis.

Ordinarily the disease is clinically well-marked at the end of twelve to eighteen hours and may at times be definitely diagnosed within four to six hours of the initial pain. It is well to remember that appendicitis is the commonest source of acute abdominal pain associated with nausea, constipation, and localized tenderness occurring in young adults. In every case of abdominal pain at least have the appendix in mind.

At the beginning of an attack of appendicitis no prediction can be made as to the final outcome, whether it will subside within a few hours or go on to a fatal sepsis. It is very evident, therefore, that if the diagnosis is made within the first twenty-four hours and the appendix removed, the possibilities of the more serious and dangerous complications have been at once removed. If, however, operation is delayed until unmistakable signs of inflammation are present, the disease may have progressed to a dangerous point before surgery is undertaken. On just what evidence, then, should we be ready to advise operation?

I believe that a great majority of conservative surgeons would consider that the good accomplished in a large number of cases by early operation while the infection is still limited to the appendix, outweighs the harm done in the few cases in which the diagnosis cannot be confirmed.

As long as there is no means of knowing early in a probable attack whether it will progress to perforation or subside within a few hours, the safest course is to advise operation as early as possible on the basis of a reasonably certain diagnosis.

It is possible to have a ruptured appendix within less than twenty-four hours of the original attack of pain. The doctor may not be called until a general peritonitis is developing. The picture then changes, but the history of the case should be a help. The location of the appendix, as has already been stated, is not always the same and the symptoms may vary somewhat on this account. As a general rule the maximum tenderness and muscle spasm are over the most inflamed part of the appendix which is ordinarily the distal end wherever it may be located. This holds good for the extracolic and retrocolic cases where the appendix is located outside or behind the cecum and the tenderness will be in the flank, loin, or costovertebral region. It may be up under the liver in which case the tenderness and pain will be higher than usual and may simulate gallbladder disease. It is not uncommon for the end of a long appendix to slip into the pelvis. When it becomes inflamed in that region, it may simulate salpingitis or it may cause bladder irritation and possibly retention. Pelvic and rectal examination will be of value in many doubtful cases. X-ray is inconclusive and may be dangerous by causing distention of a gangrenous bowel, or actually breaking through a friable appendix by the barium enema. Examination of the urine offers no evidence for or against appendicitis. The white blood count, while it may add confirmatory evidence, is in itself inconclusive.

Other conditions to be borne in mind while considering the appendix are pneumonia, typhoid fever, gastro-intestinal disorders, acute infections, lead colic, tabes dorsalis, renal calculus or infection, perinephritic abscess, acute pelvic infection, perforation of duodenal or gastric ulcer, acute cholecystitis, intestinal obstruction, peritonitis from other causes than appendicitis, torsion of an ovarian cyst, and extrauterine pregnancy. If the symptoms have not been masked by morphine, if the history is taken in its chronological order of the appearance of the symptoms, and a careful examination made, a differential diagnosis will be made in most of the cases. Occasionally there will be two horns

to the dilemma, as in a question between appendicitis and intestinal obstruction, and in such cases laparotomy will be indicated in any event and the sooner the better.

There are contraindications for operating in certain cases and chief among these are cardiac insufficiency, nephritis, and acute respiratory infections. When these are present, a careful appraisal of the two conditions must be made and the one presenting the greater danger to life must take precedence and here the choice of anesthetic is important. Of course, each individual case must be considered on its merits.

Of chronic appendicitis I know nothing. I believe the symptoms are vague, never twice alike, and the treatment effective or otherwise according to the temperamental reaction of the patient. In acute appendicitis, however, I believe that the symptoms are few, clear cut in the beginning, and definite. I believe too, that the treatment in every case in which it is possible to make a reasonably sure diagnosis should be the removal of the appendix at the earliest possible moment after the diagnosis has been made. Less than 1% of the clean cases die. More than 21% of the cases with spreading peritonitis die. From this latter group comes our high mortality in appendicitis. Much of it is avoidable.

Do not give laxatives. Do not give food. Do not give morphine. Get them operated early.

Discussion

Dr. Alexander A. Beaton, Franklin: I think this subject has been very thoroughly covered, and there is not much left for discussion. I think Dr. Woodman is to be congratulated on the amount of valuable information he has put into so short a paper.

I consider the appendix to be the most elusive organ in the body. You can find it in any shape or form. You can find it in many locations. Its most frequent site we all know but I have found it on the left side, in the pelvis, up under the liver and in a good many places where it ought not to be.

The methods of treatment are various and differ with a good many operators, and I think that the chief consideration in appendicitis is the diagnosis. Many do not recognize it at its onset, the surgeon is called later, and it may be quite a few hours more before he operates, whereas had he seen it in the early stages he could have recognized it and known just when and how to operate.

The practice of giving sedatives is to be condemned. You lose control of your patient, and the case is apt to terminate fatally because he will absolutely refuse an operation if he is not suffering.

The question of operation, and when to operate, I believe should be answered by the statement, just as soon as you make your diagnosis; but the ordinary practitioner today who advises operation is handicapped by the common knowledge of appendicitis with the feeling that every pain in the belly and every sore belly must be opened. People dread operations, and they are afraid that the appendix is often removed unnecessarily, and we all know that it has been sacrificed a great many times as a result of improper diagnosis, also that a good many normal appendices have been removed.

Now the question is, what was the cause of the pain? No diagnosis has been made in a given case. I believe in early treatment, as the doctor has said. The matter of laxatives should be carefully avoided, as well as the use of enemas, which is a common procedure in the effort to start what nature has stopped. Nature has stopped peristalsis and has blocked up the bowels, and we undertake to start this activity with enemas. I remember one case where, after I had opened it up, I found there were four quarts of water in the peritoneal cavity. The appendix had sloughed off and all the water went up through and was in the peritoneal cavity.

I have found a great number of things in the appendix. It is a catch-all for many substances including a lath nail and pieces of rubber which children had gnawed from the end of lead pencils. I have seen particles of fingernails and other things caught in it on account of its dependent position.

In cases of delayed operations the question of drainage must be considered. I do not believe in all that has been said about drainage—I mean what is generally talked of today and generally advocated. One of the recent methods advocated is that of simply leaving the abdominal wall open and putting a rubber drain down through. I think that is a failure. I do not believe in making several incisions for drainage. I believe in draining through the incision in whatever direction you are obliged to drain and then removing one drain at a time, covering a period of two or three days or more.

The symptoms of appendicitis are very illusive and deceive the best men at times. After three or four hours of severe pain in the region of the appendix I consider it unwise to delay operation. I have operated on a case three hours after the onset of pain and found a ruptured appendix and its contents in the peritoneal cavity.

I am thoroughly convinced that purgatives, enemas and opiates should have no place in the treatment of acute appendicitis excepting the use of the latter after the decision for operation has been made.

Dr. A. C. Johnston, Gorham: The last discusser is very much against the use of enemas. All cases of appendicitis, or very nearly all of them, have constipation. On the other hand, prolonged constipation usually causes pain in the colon, and how can we make a diagnosis if we do not empty the colon? I do not remember my phys-

iology very well but I do not believe we get any peristalsis above the colon due to an enema. I think it is a very good diagnostic aid to give an enema, to rule out constipation with colic pain. I examine all the appendices up in my part of the country, in the hospital there, I do not know the exact number, 200 or so last year, and, sad to say, a number of them were rather devoid of any pathology.

A MEMBER: There are two points I should like to bring out, and I am not sure that Dr. Woodman mentioned either one of them particularly. One is the mistake in diagnosing pyelitis on the right side as appendicitis. This condition is met in pregnant women, and I should like to sound a note of warning that, in addition to making very sure you are over McBurney's point in the right iliac region, you slip your hand under the right side and make pressure upward over the right kidney. Oftentimes a woman might be saved from the knife if you found this tenderness. You might properly wait awhile.

Another condition is in menstruating girls; they have quite often menstrual pains referred to the right side, and I have been confused by this condition and think I am not alone. We have put too much stress on the blood count and have found that we have taken out a normal appendix because of menstrual pains. We started in doing blood counts because of this accident on our nurses, after a good many had volunteered, and it was a very common thing to find that a menstruating girl was running a leukocytosis from 11,000 to 13,000.

I mention these two things as a warning.

DR. GEORGE S. FOSTER, Manchester: I was very much interested in the mortality rate that was taken up by the reader of this paper. Of course, we do not expect a death-rate in appendicitis from clean cases. All death-rates in appendicitis as a rule must come from unclean cases—with rare exceptions, of course. It occurred to me that sometimes in the case of a gangrenous or ruptured appendix, the case where we have either localized or generalized peritonitis, we can do too much at the time of operation. The thing to do to lower the death-rate would seem to be to get in a good drainage sufficiently deep and definite to lower the toxic condition and in that way save a life. I think we can do too much at the time of operation in septic cases.

I also feel that the postoperative treatment has a great deal to do with saving life in an appendix case that is unclean: the posture of the patient, assistance in fortifying the abdominal wall by the use of some heat, preferably boric acid fomentations because they increase drainage, the use of glucose given in some form, and supporting the patient by some cardiac or general stimulation. I think postoperative treatment in unclean appendices, and that is, of course, where the high mortality rate remains, does in a good many instances save lives and would lower the mortality if more stress, or more emphasis were laid upon these few simple points.

We could also lower the mortality from unclean appendix cases by making a stronger effort to have the appendix removed when the diagnosis was made as a clean case. An early diagnosis and an early operation would seem to be the answer.

I still believe in the white count. I agree that a blood count that shows a high white cell content is a physiological entity which comes intermittently, especially in females; the white cell count which gives the high polynuclear percentage, is the important consideration.

DR. JOHN F. GILE, Hanover: Dr. Woodman has covered the case of the acute appendix quite adequately. It is rather a smirch on the progress of surgery when the mortality figures he gives are carefully considered. We all know that too many appendices are seen that have gone altogether too far, because of someone's mistake. That error may be due to a member of the family who has injudiciously given castor oil or not called the doctor. Early appendicitis, as has been shown, may be very atypical; but as was said in the paper the keeping of the possibility of the condition in mind is to half make the diagnosis. Whether a case is going to quiet down seems to be in the hands of the gods and one person's guess, layman's or doctor's, is about as good as another's. After the diagnosis is fairly assured, the sooner the case is operated on the safer is a patient. Any acute abdominal condition which lasts longer than six hours usually means more than acute gastritis or enteritis unless there is a very definite basis such as food indiscretion. If after this length of time there is still doubt in one's mind, a consultation will avert many needless deaths. A very good illustration of this occurred last evening: A woman, aged thirty, was seen at 6 A.M. yesterday morning having vague epigastric distress. There was a history of previous similar attacks which had not been considered appendicitis. At noontime there was little change in her condition and arrangements were made for hospitalization. At three-thirty, on arrival at the hospital, there was considerable right rectus spasm but no tenderness. Temperature was 99.4, pulse 90, white count 12,000. Operation was definitely refused by the patient at that time. At 6 P.M. the patient was first conscious of pain. This was rather severe, and localized in the right lower quadrant. The right rectus muscle was hard and the tenderness was very marked. Operation disclosed a large appendix, plastered with exudate.

DR. LAWRENCE R. HAZZARD, Portsmouth: I was very glad when I looked over the program to see that we were going to discuss two subjects this morning with which we have a great deal to

do. It seems that a great many times in our medical meetings papers are read on some rare disease that is infrequently and sometimes never seen. I think we need to discuss the diseases we see most often as they cause the most trouble. I am therefore glad we have a paper on appendicitis. Moreover, it gives me a chance to talk and that makes me feel a little better. I dislike to hear a paper and not know anything about it when I should like to be able to say something.

I divide appendicitis into two phases. There is what I consider the first and second. In the first you have involvement of the appendix without invasion of the peritoneum, and when there is only involvement of the appendix, there is generalized pain over the abdomen, nausea and vomiting. In the second phase with involvement of the peritoneum there is localized pain and tenderness in the right lower quadrant. This is important in one particular type of appendicitis which I do not believe has been mentioned, and that is where the appendix is retroperitoneal, whether it lies in the iliac fossa, or directly back of the cecum with no peritoneal covering of the appendix. Because in the first place you have the symptoms the doctor mentioned, that is, in the first 24 hours there are the typical symptoms of appendicitis and then after that you may not, especially if the appendix lies posterior to the cecum, and the cecum is ballooned out, pressure on McBurney's point may not give tenderness at all, and yet at operation you may find a gangrenous appendix.

In the above condition there is tenderness in the loin. There is not tenderness in the right lower quadrant because the cecum is ballooned out and the appendix lies deep. Also in the fat abdomen there may not be tenderness but palpation in the loin will elicit severe pain. Some mention was made of the fact that it might be pyelitis but I think that can easily be ruled out by examination of the urine.

I believe Doctor Woodman said that the highest mortality was in the cases of spreading peritonitis. It seems to me it is human nature to swing one way and then the opposite way. We are doing that, for instance, with prohibition and now we are trying to reach a middle ground. I think one reason our mortality is as high as it is today, and the reason we have not made any improvement is because at one time we thought the right thing to do was to operate as soon as diagnosis was made. Ochsner advised a new method of treatment and many gravitated to his treatment because it was easy.

There are many differences of opinion in treating appendicitis. There is no doubt that men are better trained surgically, and their technique better today than 20 years ago. Then why is our mortality as high as it is? It must be the surgical judgment as to when to operate.

I believe there are definite symptoms which could be followed; a patient that has a dry skin, scanty urine, albumin in the urine, and distended abdomen is doing all he can to meet the spreading peritonitis and an operation at once is liable to result in a mortality. Glucose and intravenous saline should be given to strengthen the life that is in the balance, when immediate surgical procedure may sacrifice a life for want of careful treatment for 24 or 48 hours or even longer to get the patient into a condition where it is safer to operate.

Dr. Paul Dye, Wolfeboro: I have recently been in contact with Dr. Eugene Pool's Surgical Service at the New York Hospital, New York City, and served for 26 months on Dr. E. P. Richardson's Surgical Service at the Massachusetts General Hospital in Boston. I believe Dr. Woodman has presented a very excellent and an unusually comprehensive paper, considering the scope and importance of the subject, in the short time allotted him.

The mortality rate in cases of ruptured appendix can be lowered by careful, suitable postoperative treatment as well as by the skill of the surgical procedure at the time of operation. In these cases, all efforts possible to limit intestinal peristalsis should be made; such consisting of prohibiting any food or drink by mouth, and frequent hypodermics of morphia. The morphia aids by abolishing pain thus inducing restfulness in the patient, and likewise directly limits or inhibits intestinal peristalsis.

Fowler's position in bed to secure gravity drainage of septic material is important and valuable, for elimination of such material as well as to aid the localizing and walling-off processes in the pelvis.

Supportive measures to combat dehydration and to supply easily assimilable food in the form of hypodermoclyses of 2½% glucose in normal saline solution should always be carried out; particularly as long as the fluids are contraindicated or limited by mouth ingestion.

A great many men have been giving saline and glucose solutions by rectum. Recent experimental evidence shows that this procedure is practically valueless. Very little fluid or glucose is absorbed through the rectal mucosa during the first 24 hours; none thereafter. Furthermore, rectal taps become, in time, irritating and discomforting to a patient who is most urgently in need of absolute rest and quiet.

Dr. D. W. Parker, Manchester: There is very little that I can add to Dr. Woodman's discussion. There are, however, several points that he has touched upon which might be stressed further. We were led to believe, while in the Medical School, that appendicitis exhibited a rather definite stereotyped train of symptoms, namely, epigastric pain followed by nausea and vomiting, and later, a shifting of the pain to the region of the appendix, fever, and leukocytosis. I think most of you will agree with me that appendicitis does not always follow such a clearcut picture.

As to treatment, I am firmly convinced that the appendix should be removed immediately on a reasonably sure diagnosis. I do not say positive diagnosis. The importance of early removal of an acutely inflamed appendix distinctly outweighs the possible danger of going into an abdomen for a supposed pathological appendix which may not exist.

I wish to emphasize what Dr. Woodman said in regard to rectal examinations, particularly in the case of children. Not infrequently we see a child in the hospital or at home with symptoms which are consistent with appendicitis, but rigidity over the appendix may not be present to any appreciable extent or not at all. Sometimes tenderness is not appreciably marked In these cases, the appendix may lie in the pelvis, and a rectal examination will demonstrate the condition definitely. The importance of rectal examination cannot be over-emphasized. It should be carried out as a routine measure in every case where the diagnosis is at all doubtful. I am sure that definite information will be obtained many times by this procedure where it could not be found in any other way.

In regard to leukocytosis, I agree with Dr. Woodman that it is valuable if present, but unfortunately, some of the worst cases are not accompanied by a definite increase in the white count. It is my belief, also, that the differential count, many times, gives more valuable information than the total count.

As to drainage, my custom is to drain in all doubtful cases. I do not believe that it does any definite harm. Drainage may be introduced through the incision, or through a stab wound. Where there is free pus in the abdomen, the drainage should be adequate. My custom is to use two or three rubber tube drains, one to the bottom of the pelvis, another outside the ascending colon to the right kidney fossa. If drainage is introduced through a right rectus incision, there is always the possibility that the deep epigastric artery may be eroded from pressure of the tubes. When this occurs, a very sharp hemorrhage may take place. I, personally, have seen this occur on several occasions. I believe that it is good surgery to ligate the deep epigastric artery at the time of operation in the pus cases where drainage tubes must be introduced through a right rectus incision.

Dr. Thomas W. Luce, Portsmouth: Dr. Woodman gave us, as he called it, an "excuse" for his paper by his recognition of the increase in mortality. He did not separate those cases that die so far as he might have, but it was apparent that the rate is increasing, and his hope is in some way to decrease that rate. I had a recent experience which seems to suggest some constructive possibilities along this line in New Hampshire.

I operated on a young boy three months ago for an acute appendicitis in the middle of the night, and the story in connection with that case was this. This boy, living in York, Maine, went home from school with a pain in his abdomen. His mother started for the castor oil bottle but the younger sister said, "Mother, you must not do it. Our teacher says you must not give physic, when there is pain in the abdomen. You must call the doctor." This was done, and the boy was operated on. This was in Maine, and I learned that is the policy there in the schools to have the teacher give this instruction to the children. It is pretty hard to get to the parents, to talk about this danger of cathartics, which we recognize in appendicitis, but in Maine the children in the public schools are told by the teacher, and in this case it did a great deal of good.

I wonder if something of that sort could not be done in our state. I can see in this plan the possibility of cutting down our mortality in appendicitis, and I think patients often have had this bad treatment before the doctor sees them. I do not think we operate less skillfully, or that the general physician in the state of New Hampshire is slower than he used to be, but I think people do more doctoring at home than formerly, and the doctor sees the case sometimes when it is too late to give the patient the benefit of an early operation—the Maine plan will prevent this in some cases.

Dr. J. C. Tappan, Derry: There is one point I have not heard mentioned in the differential diagnosis. It occurred in one of the cases I had. That is the importance of a differential diagnosis between appendicitis and infantile paralysis. I had a case which in every way resembled a case of appendicitis but which unfortunately turned out to be a case of poliomyelitis with paralysis of the lungs, followed by death. It was before the use of the Drinker inhalator and nothing could be done for the patient in any way. But this was a case which appeared to be appendicitis and turned out to be infantile paralysis.

Dr. Woodman, closing: Of course, if you operate on a large enough number of cases of normal appendices you will reduce your mortality figures, but that was not the burden of my discourse. I am glad someone spoke of Ochsner's treatment. Personally, I never know which cases are going to fall into the Ochsner group, that is, which ones are going to get walled off and form that nice, round, hard ball which you can drain at your leisure, because I am always afraid they are going to get in the class of spreading peritonitis. On the other hand, there is something to be said on that subject. If you see your patient after he has been ill for several days and he is forming a pretty well walled off appendix, and is in a pretty bad condition, that no doubt is worth considering.

I think that the rule stated in the paper will

probably save just as many lives, however, as the Ochsner method if you stick to it over a period of time.

Dr. Johnston spoke of enemas and peristalsis. I was not trying to prove that an enema sets up peristalsis that did the damage. I am not afraid to give a small, low enema, but I would warn anybody against giving the kind of enema that Dr. Beaton pointed out. I happened to know about that case, and that was an enema, plus. It was an enema with a vengeance. It did not stop as a low enema but it shot right up on through the perforation and filled the abdominal cavity, and in that case it did not do any good.

Pyelitis was spoken of and examination of the lower costal region. Dr. Foster spoke of the death-rate, which he said should be zero. It is a fact in one sense, and I think that fractional percentage of mortality is due not necessarily to those factors but to the fact that all surgical cases in some small percentage are victims of postoperative pneumonia and various other complications. Of course, occasionally a case will become infected even though it is clean. Those cases are the ones that give you your small death-rate in the clean cases.

I want to thank Dr. Dye for supplementing my postoperative treatment. I believe it to be absolutely correct to put drained cases in the Fowler position, and glucose and saline are indicated often.

INVERSION OF THE UTERUS*

With Report of An Unusual Case

BY EMERY M. FITCH, M.D.,† CLAREMONT

INVERSION of the uterus, although recognized as far back as A.D. 110, when Soranus described "inversio uteri", is of fairly rare occurrence; so rare, in fact, that many obstetricians with a large practice have never personally observed a complete inversion.

In studying the literature of inversion of the uterus, one notes wide variance of opinion as to the frequency of occurrence, the etiology, treatment and prognosis. Findley found in a study made in 1929, one case to 113,068 labors. Others observe one to 40,000, and some believe it occurs as often as one to 23,000.

Inversion of the uterus may be partial or complete. In the partial state there is an indentation of the fundus to a varying degree, but the body does not pass through the external os; while in the complete type the fundus goes through the external os, the cavity of the uterus becomes the outside and the peritoneal portion becomes a cup leading up to the pelvic cavity.

The partial is without doubt of fairly frequent occurrence in obstetrical practice but is of so slight severity that it rights itself without recognition, excepting for a severe but short hemorrhage immediately after the placenta is delivered. The uterine sinuses are held open for a time by too active Credé manipulation, but when the fundus is relieved of the external pressure the indentation becomes forced outward by the formation of a clot, the uterine muscle begins to contract normally, and the condition is cured spontaneously. The greater the extent of the partial indentation, the more easily it becomes converted into the complete type, for the indented portion, when caught by the ring of contracting muscle, is forced through the inactive lower uterine segment and the process rapidly goes on to a complete inversion.

All inversions are at first acute and, if not relieved, are converted into the chronic condition by a change in the uterine tissue which takes place at a variable length of time; usually within twenty-four hours from the onset. This change chiefly consists in the formation of a contracting ring, either at the site of the internal os, or, more probably, Bandl's ring, which will not allow the fundus to be pushed back through no matter how persistently or in what direction force may be applied.

The cause of acute uterine inversion is invariably puerperal and can nearly always be traced to some slip in the technic of delivery. Chief among these mistakes is traction on the cord or forcible expression of the placenta by the Credé method between pains, or a combination of the two. However, inversion has been known to occur when the placenta was delivered spontaneously, without any assistance. Fundal attachment of the placenta is supposed to be a large contributing cause, yet cases of inversion have been reported in placenta previa. The majority of cases occurred in primiparae, but the accident has taken place in multiparae whose previous labors have all been normal.

Atony evidently plays an important part. Especially is this true when there is atony of the lower uterine segment and contraction of the fundus. Pressure externally on the fundus, even during contraction, accompanied by only a slight pull from below may start invagination of the relaxed lower segment, and the active uterine contractions of the fundus may complete the inversion.

After any portion of the uterus becomes indented to a considerable extent, the rest of the organ seizes this invaginated portion as it would grasp a foreign body, and in attempting to expel it, turns itself inside out. Without doubt this factor also works in the other direction and often brings about spontaneous cure of a small partial inversion.

*Read at the Annual Meeting of the New Hampshire Medical Society at Manchester, May 17, 1932.

†Fitch—Surgeon, Claremont General Hospital. For record and address of author see "This Week's Issue," page 429.

There are cases on record of the non-pregnant uterus becoming inverted when nature tried to expel a tumor, either a mucous polyp of large size or a submucous fibroid of the fundus. These cases, however, are extremely rare.

Acute inversion is marked by sharp pain, sudden severe hemorrhage, shock, and collapse. The pain, however, may be absent, for the inversion oftentimes takes place while the patient is still under a general anesthetic.

Chronic inversion may give very few symptoms. The accompanying hemorrhage, which is usually the chief symptom, may be only occasional, resulting in increasing anemia. The extreme malposition of the bladder leads to urinary disorders, and the presence of the tumefaction in the vagina produces rectal symptoms.

The treatment of acute inversion should be guided by the amount of shock and hemorrhage. If shock is not too pronounced the uterus should be immediately reinverted. This can be accomplished better by pressure along the sides of the uterus than by direct pressure on the fundus.

If hemorrhage is profuse the mass in the vagina should be compressed manually and the bleeding controlled by the operator's fingers before attempting to correct the inversion. If shock is pronounced the vagina should be firmly packed with gauze and attempts at reinversion delayed until the patient is put in better condition by blood transfusion or intravenous injections of normal sodium chloride solution. After the patient has recovered from shock, reduction can be performed or the packing continued until the patient has regained strength, the inversion being allowed to become chronic and then treated surgically.

There is no great glory in restoring an inversion immediately if the accompanying shock is going to mean the death of the patient. There is far less danger of sepsis in allowing the condition to become chronic than there is in replacing a badly traumatized uterus.

In chronic inversion of the uterus the contraction of the cervix is such that manipulation from below fails in producing a cure because the cervical contraction ring prevents the swollen, thickened, and inverted fundus from being forced up through to its normal position until this contracting ring is stretched or cut.

The cure then invariably demands some form of operative procedure. The particular kind of operation depends upon the individual skill of the operator or upon the local objective symptoms. Most surgeons prefer the vaginal operations, either anterior colpohysterotomy or hysterectomy, although some adhere equally firmly to the abdominal operation, finding it more satisfactory to reinvert the uterus by pulling up on the cornua from above. The adherents of the vaginal operations object to the abdominal route on the ground that it is more liable to lead to general abdominal infection. Sometimes the vaginal operations are rendered impossible by excessive swelling of the vulva and vagina.

Of the vaginal operations, the one most suitable is that of anterior colpohysterotomy, the so-called Spinelli operation.

Patients who have had an operation for chronic inversion of the uterus and subsequently become pregnant should be treated the same as those who have had a previous classical cesarean operation. That is to say, they are liable to rupture of the uterus during labor and should have a cesarean operation at term, or at least should be watched very carefully during labor with every possible means at hand to do a quick cesarean if the labor is at all difficult.

REPORT OF THE CASE

Mrs. F. O. Aged 25. White, married, Protestant. Occupation housewife. Admitted to hospital service by Dr. Brooks March 25, 1930.

Family History: Mother living and well. Father died of pneumonia. Has three brothers and three sisters, all living and well.

Personal History: Has had most of the children's diseases. Had pneumonia in 1925 with good recovery and no permanent after-affects. No other sickness. Began menstruating at fifteen. Has always been more or less irregular. Periods usually last eight days with a medium amount of flow accompanied by a great deal of pain. Has one living child six years of age that was a forceps delivery, the baby weighing 9 pounds. Since then has had three miscarriages, each one at the sixth week of gestation.

Present Illness: Full term pregnant condition. Has been very constipated throughout pregnancy. Troubled greatly with insomnia; appetite poor. Three months ago began having false labor pains and was admitted to the hospital for this condition on three different occasions. Admitted March 25 in labor stating that the membranes had ruptured several days ago.

Physical Examination: General appearance that of a very anemic pregnant woman. Head and neck normal, no enlargement of thyroid. Chest normal. Heart tones clear and regular. Pulse 100, B. P. 140/78. Lungs clear front and back. Breasts soft, no masses. Bones and joints normal. Abdomen negative except for gravid full term uterus. Pelvimetry shows slight flattening of pelvis. No varicosities.

Delivery: Labor persisted fairly active for eight hours, but owing to lack of amniotic fluid, dilatation was slow and incomplete. At this time Dr. Keeley was called in consultation, full ether anesthesia given, dilatation completed manually, and mid-forceps applied, resulting in delivery of a normal child weighing 7½ pounds. The cervix was slightly torn. The placenta was delivered by Credé after a wait of 10 minutes.

Immediately after the delivery of the placenta there was severe hemorrhage, and examination showed that the fundus of the uterus had become inverted and delivered through the torn cervix, resulting in a complete inversion of the uterus. The fundus was with difficulty reinverted through the cervix to its normal position, the cervical tear sutured with catgut, and the vagina packed with a sterile towel.

Postpartum hemorrhage was fairly easily controlled, and the patient made an average recovery, being discharged from the hospital on the seven-

teenth day after delivery. At this time pelvic examination showed the cervix soft and patulous, fundus in good position and normal in size for that state of the puerperium. Patient was still very anemic and consequently was discharged with instruction to see her physician weekly.

Two weeks after leaving the hospital, the patient, while doing some work about the house, was suddenly seized with severe pains in the pelvis accompanied by a gush of blood from the vagina and extreme prostration. Dr. Brooks was immediately called. Examination showed that the uterus had again become completely inverted. The vagina was thoroughly packed with gauze and the hemorrhage controlled. The patient was kept in bed for one week with vaginal tamponing, which partially controlled the bleeding.

On May 2, 1930, being five weeks after the first accident and one week after the recurrence, she was again admitted to the hospital, and I was called upon to treat her surgically.

At this time the patient was still nursing her baby. The anemia was pronounced, the skin pale and waxy. Blood pressure 138-78. Temperature 98.8°. Pulse 80. Hemoglobin 29%, coagulation time six minutes. Erythrocytes 3,170,000. Leukocytes 5,700. Vaginal examination showed a pear-shaped tumor in the vagina, gray in color until touched, when it bled easily and profusely. This tumor proved to be a completely inverted uterus, coming through the cervix, filling the vagina, and extending to the vulva. Operation by vaginal route was advised.

Owing to the extremely poor condition of the patient, blood transfusion was considered, but abandoned after three donors had been tried resulting in incomplete matching.

May 3, 1930, operated under gas oxygen and ether anesthesia. Sterilization of vagina and uterine tumor was accomplished with tincture of iodine. Vaginal specula introduced. The cervix was grasped with four volsella forceps and held while careful pressure was made on the fundus and sides of the uterus with an attempt to deliver the inversion back through the cervix. This procedure failed, however, because of a firmly contracted ring in the region of the internal os.

A transverse incision was made on the anterior vault of the vagina in front of the cervix. A sound was introduced into the urethra to give the position of the bladder. By blunt dissection the bladder was elevated. The bulging peritoneum was then cut in a transverse direction, thus admitting the finger to the pelvic cavity. With a scalpel a cut was then made through the anterior lip of the cervix and the anterior wall of the uterus, well up to the top of the fundus, in order to prevent tearing when replacement of the inversion was attempted.

It is interesting to note in this connection that the musculature of the uterus is such that, having been turned inside out and left in that position and pressed upon by packing material, the organ is restored to its normal position with a considerable amount of difficulty.

Having been reinverted, the uterine wall was sutured with two layers of chromic catgut, the innermost layer being buried in the muscle well down to, but not involving, the mucosa, the outer layer closing the peritoneal edges. The fundus was then replaced within the peritoneal cavity, the opening in the vagina was closed with chromic catgut and the vagina packed with gauze. Hypodermoclysis of 1500 c.c. normal salt solution was given.

Patient made an uneventful recovery for eleven days. On the twelfth day she was taken with chills and the temperature rose to 104.5°, the pulse from 80 to 110. She had repeated chills, some abdominal tenderness, and rather marked delirium, with a temperature curve characteristic of sepsis for three days, when the symptoms all cleared up and she returned to normal. The baby was taken from the breast the day of operation and put on a formula. The breasts gave no trouble.

The patient was discharged from the hospital on the nineteenth day following operation. She has recovered from her anemia, is now well after a lapse of two years, and has not as yet become pregnant.

The interesting feature of this case is recurrence of the inversion, which had been replaced in the acute stage and had remained in place as long as the patient was quiet in bed. Evidently the pronounced anemia and general feeble condition of the patient were material factors in the second accident.

REFERENCES

1 Friedman, L. V.: Inversion of the uterus, with report of two cases replaced by laparotomy. J. A. M. A. 45:1920 (Dec. 23) 1905.
2 Peterson, Reuben: Inversion of the Uterus. Nelson Living Surg. 7:697.
3 Haselhorst, G.: Life-saving effect of blood transfusion in case of complete inversion of the puerperal uterus. Zentralbl. f. Gynäk. 51:910 (Apr. 9) 1927.
4 Findley, Palmer: Acute puerperal inversion of the uterus. Am. J. Obst. & Gynec. 18:587 (Oct.) 1929.
5 McCullagh, W. McK. H.: Inversion of the Uterus: a report on three cases and an analysis of 233 recently recorded cases. J. Obst. & Gynec. Brit. Emp. 32:280, 1925.
6 Irving, Frederick C., and Kellogg, Foster S.: Two additional cases of acute puerperal inversion of the uterus treated by abdominal replacement. Am. J. Obst. & Gynec. 22:440 (Sept.) 1931.

Discussion

Dr. Robert H. Brooks, Claremont: The etiology of this condition and the treatment, after it comes to the care of the surgeon, were so completely taken up in the previous paper that there is no occasion to add more.

This individual case, however, from the beginning of her third stage to the day of her operation presents certain details that are instructive, and may be of assistance to us in case a similar accident happens in our future practice of obstetrics. Edgar says in one place, "Mismanagement is generally responsible for this complication," and in another, "In very rare cases the complication may exist without reproach to the physician." It is not the intention in this discussion to establish any alibis or to present excuses or explanations, only to offer the facts as we found them.

The patient came to delivery in poor physical condition, she was undernourished, had been living in external conditions of poverty, and she had a marked degree of secondary anemia, so it was quite the usual thing that she should develop uterine inertia after trying eight hours to dilate the cervix without the benefit of the bag of waters. With the handicap of a shortened anteroposterior diameter of the outlet, relief by forceps seemed clearly indicated.

It is true that the placenta was delivered by the Credé method, plus traction on the cord, but the patient was recovering from her anesthetic, pressure on the fundus was during a contraction, traction on the cord was not undue; on these points my consultant and I are positive. Things had been progressing smoothly, the pa-

tient was not flowing in excess, there was no hurry.

After the placenta, came the deluge, and very soon a true knowledge of the conditions. Through what proved to be a deep cervical tear the uterus had inverted and we had the inside of the uterine cavity presenting at the vulva. Traction on the edges of the cervix plus pressure on the inverted fundus were successful in reducing the condition, and then the cervical tear was discovered. This seems to me to be the main cause of the accident, and the first point I hope to emphasize and from which we may learn, is that a tired uterus, contracting in uneven bands plus even normal pressure on the fundus and slight traction on the cord may, in the presence of a cervical tear, invert easily.

The second point to be developed can be supported only by theories, and perhaps further discussion may establish the facts. This patient went back to bed in severe shock from hemorrhage and from uterine manipulations, but after a somewhat troublesome convalescence was discharged well, except for the anemia, for which she was kept under treatment. We knew the cervix had been properly repaired, we believed the fundus was in normal position when she returned from the delivery room, and during her convalescence the uterus went through normal subinvolution and was normal on discharge, so far as our examinations could determine. However, some days after she got home, when lifting a heavy weight she felt a sudden sharp pain and had a considerable showing of fresh blood. Examination shortly after revealed the inverted fundus in the vagina. Theory only can be advanced to account for this second inversion. With the pale toneless condition of all muscles in secondary anemia it requires little imagination to see the abdominal muscles soft and lax, the bowels a dead weight pressing on the fundus of a uterus that had become soft and boggy or contracted in uneven bands. Sudden lifting with the line of endeavor in the same plane as the cavity, and inversion occurs again. At subsequent operation the cervix was found healed but soft and patulous.

This is offered as a possible theory to explain the second inversion, more in the hope that your further discussions will advance other theories than in the belief that this is the correct solution.

Dr. Charles F. Keeley, Claremont: Dr. Brooks mentioned that, at the time of the delivery of this woman, she was in a very anemic condition, which, no doubt, was an important factor in causing it; and, added to this, we have the three necessary factors which were present for the making of the inversion of the uterus, namely, thinness and loss of tone of the body of the uterus, cervical laceration which rendered the cervical canal more patulous, and Credé of the fundus of the uterus.

I fully dilated the cervix before forceps were applied and the delivery was not at all difficult.

As soon as an inversion of the uterus was manifested by the profuse hemorrhage and a tumor in the vagina, I returned the fundus of the uterus into its normal position and sutured the cervical laceration, which was not extreme. The vagina was packed and the patient was returned to bed. At the time of the patient's discharge from the hospital, vaginal examination by Dr. Brooks showed the uterus to be in its normal condition.

The question is, What caused a return of the inversion? In regard to this we can only theorize.

First, possibly a small indentation of the fundus existed and the anemic condition of the woman was cause of loss of tone.

Secondly, the cervix was still relaxed and the muscle of the uterus was grasping on the indentation, contracting, trying to expel it as a foreign body, and with the added pressure, which came on with the woman on her feet and about the home, completed the return of the inversion necessitating further surgical treatment as described by Dr. Fitch.

It is very fortunate indeed that this condition is rare both for the sake of the patient and the attending obstetrician. From the point of the patient, mortality runs as high as 50% because of the added complications of strangulation, the resulting necrosis of the uterus and the uterine sinus opened into an infected cavity made worse by trauma. From the standpoint of the obstetrician, the difficulty is in returning the fundus in the acute stage.

Dr. Chancey Adams, Concord: I was much interested in this case because, like Dr. Fitch, I have had only one case, and if you will permit, I will read from my notes.

This woman, aged 23, was married in December, 1920. Her baby was born December 26, 1921. The labor was hard, with forceps delivery, under ether. The baby weighed eight and one quarter pounds. The patient did not nurse it. She was referred to me on May 1, 1922, by Dr. Arthur J. Rowe of Penacook, who informed me that before he left the case, after delivery, the uterus had contracted and could be felt contracted above the pubes. Four months and ten days after the birth of the baby she was referred to me. About three weeks before, she had a severe hemorrhage, and again the day before I saw her, on April 30. She had flowed constantly since the baby was born, sometimes only a little, with clots, large and small. She has had but little pain since the confinement. When I saw her the uterus could not be felt above the pubes.

On examination a small round tumor was felt in the vaginal cavity high up which I thought was a fibroid freed in the course of labor and extended through the canal by uterine contraction. On account of the patient's extreme sensitiveness and resistance, my examination was not very satisfactory. The vagina was full of clots. Under ether, however, the tumor proved to be an inverted uterus. The abdomen was then opened, Dr. D. G. McIvor assisting me, and, with the uterus grasped with both hands, fingers on the fundus and thumbs on the cervix, after repeated efforts the uterus was replaced. This was accomplished after several efforts. The

posterior wall was torn from the fundus to the internal os. The incision was properly closed. The patient made a good recovery, and was discharged well on May 17, 16 days after admission. Her convalescence was without event.

I saw her September 29, 1922, when she said she was in perfect health and menstruated regularly and normally. On April 21, 1932, 10 years after, her doctor informed me that she has enjoyed good health since her discharge from the hospital.

Dr. Fred E. Clow, Wolfeboro: It would seem scarcely possible that one could have so serious a situation as inversion of the uterus confronting him, without recognition, yet there are at least two instances in which this has occurred.

A few years ago, in the London *Lancet*, a case was reported in which a physician, following delivery of a woman, removed forcibly the uterus, one fallopian tube and one ovary, under the impression that the protruding mass was a fibroid. Even then he did not realize what had happened until a loop of small intestine presented at the vulva. Strange to say the patient did not die of shock or hemorrhage, but succumbed ten days later from peritonitis.

A patient was brought to our hospital eight months after the birth of a baby, with the diagnosis of uterine polyp. Her physician had been present at the birth and noted nothing unusual. Pelvic examination disclosed absence of the fundus. An attempt was made to reduce the inversion by the method used by Dr. Fitch in his case, and as described by Graves. The cervix was tightly gripping the upper portion of the body of the uterus. The vagina was narrow and shallow. Vaginal hysterectomy was easily performed and the patient made an uninterrupted recovery.

Dr. John J. Boardman, Hanover: I have just a word to say merely because I am one of the 113,000 who have had the unfortunate experience of having one of these cases. It so happened that when I was a young boy I saw a veterinary return an inverted uterus in a cow, and I had heard of it in the medical school just in skipping over the chapter headings. Otherwise I had never heard or seen anything about inversion of the uterus, and now when anybody speaks of it the perspiration starts in my hands and my knees get shaky, and I have a very vivid picture of a morning last June, about 5:30, when I was attending a rather close friend. She had had a normal pregnancy. She was an athletic type of woman, and everything indicated a perfectly normal confinement, in fact, everything was normal until I was delivering the placenta, but as I was removing the placenta I discovered something that felt like an orange with a blood clot coming away. I did not know what it was. I thought of everything. I felt like the woman in one report I heard who, down in Kentucky, thought it was a second child and with a little contraction delivered the uterus, an ovary and one tube and the patient lived 24 years after that. Anyway, I was fortunate to be in the hospital where I could have help, and some friends came to my aid and the uterus was successfully replaced.

I have not any comment to make on the paper. I was just wondering why Dr. Fitch made his replacement through the vagina rather than through an abdominal incision, as most of the reports that I have looked up show. I did quite a lot of studying on this question of inversion after that one case, hoping I would never see one but if I did I would know what to do. I am wondering if it would not be wise if more time were spent perhaps in the medical school in giving us young fellows a little more information about such subjects. I am thinking, if Dr. Fitch were not already grayheaded, that it would make gray hairs just having a case of inversion.

I feel like Mrs. Coolidge so far as getting more information about this condition is concerned. The story was told last week about Ex-President Coolidge who was at church when some persons came to call on him, and Mrs. Coolidge was trying to entertain them until he returned. When he did appear she said, "Well where have you been?"

He said, "Been to church."

"What did the minister talk about?"

"Talked about Heaven."

"What did he say?"

"Oh, he's in favor of it."

Dr. Paul Dye, Wolfeboro: This is not a comment or addition to the very interesting report which Dr. Fitch has given but merely for the purpose of information. I should like to know from the technical standpoint just which is the operation of choice, and how you can decide whether to do the operation that Dr. Fitch mentioned and of which he gave such a graphic description, or do a vaginal hysterectomy?

Dr. Fitch, closing: I think the choice of operation for inversion of the uterus depends wholly upon the individual surgeon. There are surgeons who always follow the abdominal route. Irving of Boston has reported several cases, all done by this method, and he makes it look very easy, by just pulling up on one cornu of the uterus, to turn it back to its normal shape and position.

I have frankly never had but one of these cases and I do not know that I will ever have an opportunity to operate on another; but in our case the organ was so firmly contracted I do not see how it could have been reinverted until it was split, and this could be best accomplished by the vaginal route.

If I ever get another one of these cases I am going to do it by the vaginal route because it is easier for me than by the abdominal, and, in addition, I think the chance of infection is much less through the vagina than if you go in through the abdomen, where you have to deal with the intestines.

The choice of operation is similar to that in

treating cancer of the cervix. There are certain surgeons who do a vaginal hysterectomy fairly easily. There are others who rarely attempt one. I find it easier in nearly all cases of complete hysterectomy to do it by the vaginal route than by either the combined or complete abdominal route. I admit it sometimes looks hard. We have up in Claremont a very honest kind of a doctor, in fact they are all honest, but this fellow came in one day while I did a vaginal hysterectomy, and looked over my shoulder to see what I was doing, and he said, "I do not believe that can be done." I said, "I do not know that it can, but I am going to keep on until I do it." He remained through the operation and was convinced, but I met him on the street in the afternoon and he came up to me with a smile and said, "I still do not believe anybody can take a uterus out through the vagina."

Regarding vaginal hysterectomy or any hysterectomy for inversion of the uterus: that should be avoided because these women are young and cases are reported where they have gone through subsequent labors without difficulty.

I think this answers Dr. Boardman's and also Dr. Dye's questions.

VERMONT STATE MEDICAL SOCIETY

VERMONT DEPARTMENT OF PUBLIC HEALTH

July, 1932

The following list of communicable diseases has been reported to this Department during the month: chicken pox 52, diphtheria 2, measles 210, mumps 215, scarlet fever 29, smallpox 2, typhoid fever 4, whooping cough 109 and undulant fever 3.

The Laboratory of Hygiene's examinations total 1,685 for the month, classified in the following manner:

Examination

For diphtheria bacilli	88
For Widal reaction of typhoid fever	40
For malarial parasites	1
For tubercle bacilli	135
For evidence of syphilis	445
For gonococci in pus	154
Of water, chemical and bacteriological	214
Of milk, market	219
Of water, bacteriological	223
Of milk, submitted for chemical only	5
Of milk, submitted for microscopical only	81
Of foods	19
Of drugs	0
For the courts, autopsies	0
For the courts, miscellaneous	5
Of animal heads for evidence of rabies	1
Miscellaneous	54
Autopsies to complete death returns	1

In the Division of Communicable Diseases, 32 new cases of gonorrhea and 29 new cases of syphilis were reported. Three hundred and eighty-seven Wassermann outfits and 152 gonorrheal slides were sent out from this office.

The Division of Poliomyelitis After-care nurses made 153 home visits in July. Two patients were admitted to the Audubon Hospital and two patients discharged from the same hospital. Twenty-nine pieces of apparatus were fitted, seven pieces of apparatus repaired and 46 orthopedic corrections made to shoes. Sales were made during the month by the vocational worker amounting to $25.00.

Child health clinics were conducted in nine towns of the state at which the nurse of the Maternity and Infancy Division coöperated. Two thousand, six hundred and thirteen pieces of literature were distributed during the month by this Division.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 18351*

A CASE OF EXTREME HYPERTENSION IN A WOMAN OF TWENTY-FOUR

MEDICAL DEPARTMENT

PRESENTATION OF CASE

DR. JAMES A. HALSTED**: This is the case of a mentally defective unmarried housemaid twenty-four years old at the time of her first entry.

At the age of eighteen she had a full term pregnancy, and another at twenty. Five years ago she had an acute gonococcal infection. At that time she had dyspnea on exertion which had gradually increased in severity. Two and a half years ago she again became pregnant and at this time had severe headaches. She had a blood pressure of 220/135 and gallop rhythm. There was a large trace of albumin in the urine. The pregnancy was interrupted and a hysterectomy was done. A year and a half ago she began having attacks of nocturnal dyspnea with wheezing, and noticed some numbness in the left shoulder. A month later she became orthopneic and had attacks of dizziness with black specks before her eyes.

First admission. Fifteen months ago she entered the hospital for the first time. The chief complaints were dyspnea and headaches. Her blood pressure was 220/135. She had a moderate amount of pyorrhea and a vaginal discharge. The heart was slightly enlarged. The first sound at the apex was reduplicated but the pulses and arteries were normal. Renal function tests at that time showed no evidence of renal insufficiency. The urinary output was normal and a Mosenthal test showed the specific gravity to be 1.030 to 1.032. There was no anemia. Fundus examination showed the disks hazy and red, the veins engorged and nicked.

She became much better after two weeks in the hospital in bed. After she was digitalized and given potassium sulphocyanate the blood pressure came down to 160/90.

History of interval. She got along well during the next year with only moderate headache and occasional dyspnea on exertion. Four months ago her symptoms had increased and she began having further attacks of nocturnal dyspnea. One week before reëntry she was nauseated, vomited and had dull constant substernal pain.

Second admission, sixteen months after her previous discharge.

Physical examination showed the heart considerably enlarged, more so than at the first admission. The left border of dullness was 4 centimeters outside the midclavicular line. At the apex there was a brief systolic murmur with a soft diastolic murmur. A third sound was audible. There was gallop rhythm and the aortic second sound was much accentuated. There was a soft systolic puff at the aortic area, with a low pitched early diastolic murmur at the left of the sternum in the third interspace. The blood pressure was 270/160 to 215/125. The liver edge was palpable 2 fingerbreadths below the costal margin and was tender. The eyegrounds showed swelling of the nerve heads, exudates and hemorrhages.

At this time the red cell count was 3,500,000, the hemoglobin 65 per cent. The urine showed a large trace of albumin and many white cells and casts, no red cells at entry, though later some were found. The non-protein nitrogen was 53, the phthalein output less than 5 per cent in fifteen minutes. One of three stool examinations showed a strongly positive guaiac.

Venesection was done with considerable relief of the headaches. On the fifth day she suddenly became very drowsy, with irregular respirations, though not of the typical Cheyne-Stokes type. She had rather thick speech and complained of not being able to see out of her left eye. The pupils did not react to light. The reflexes were normal and the neck was not stiff. Two days later she could see with both eyes. The speech was still slightly thick. She continued in this condition, very drowsy. Two days before death, ten days after this episode occurred, the non-protein nitrogen was 63. There were no convulsions. She died sixteen days after entry.

CLINICAL DISCUSSION

In Third Year Class

DR. MAURICE FREMONT-SMITH: We have a picture of a young woman with very marked hypertension and evidence of a failing vascular system, both heart and cerebral vessels.

I have never heard heart pain described as shoulder numbness, but probably this numbness in her shoulder was a symptom of angina. We often have pain running down the arm accompanied by numbness of the arm and hand, but I have never known of numbness of the shoulder.

A doubled first sound may mean nothing; on the other hand it may mean bundle branch block. Without an electrocardiogram we cannot tell

*This report gives the essentials of two discussions, one in Dr. Cabot's Third Year class, one at a conference of the hospital staff.

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positively, but when we do get a first sound doubled it is worth attention. A doubled second sound is without known significance.

If the pulses and arteries were normal there must be arteriosclerosis confined to the smaller vessels of the kidneys, the retinae and the brain. In such cases we do not find accompanying sclerosis of the larger vessels. A diastolic blood pressure of 135 is extremely high for a girl of twenty-four.

We can have in arteriosclerosis a picture in the fundus indistinguishable from that in cases of brain tumor with marked intracranial pressure. We can have a picture similar to this with failing kidneys, with uremia. We should expect some hemorrhages, some exudate. We have here simply edema of the disks, which can occur in the pre-uremic stage.

In those blood tests we have no evidence of nephritis, no backing up of nitrogen, no real anemia, which in the old days I should have said must exist with chronic nephritis, but I am learning that we can have a certain kind of nephritis of the vascular type without marked anemia. In the chronic parenchymatous nephritis we do get anemia; if we do not, we can be pretty sure we have not parenchymatous nephritis. Some nephritides however can have pretty definite evidence in the kidneys without anemia. Here she is so far without any nitrogen retention. You may ask why I say there is any evidence of nephritis at all, with good renal function, no anemia and a good output. I think, on the basis of this tremendous hypertension, high diastolic blood pressure and the albumin in the urine, we can make a diagnosis of arteriolar sclerosis involving the kidney; in other words a vascular nephritis.

I do not know what the cause of this diastolic third sound was. Of course we rather think here of a very early mitral stenosis. It is one of the first signs we get as mitral stenosis is developing. The first evidence of stenosis is a sharpened first sound and an accentuated pulmonic second sound. The first thing that actually makes us focus attention on the mitral valve is that low, rather late sound in diastole which is a characteristic mitral diastolic murmur. Here I do not know what causes it. We get a gallop rhythm, which sounds the same in a rapidly beating heart, in a heart with marked myocarditis and in a failing heart from any other cause.

I should have expected that high fixation of gravity to be associated with heart failure and not kidney disease. It is interesting that here the kidney could not dilute the urine.

We know that in a girl of this age such hypertension must be malignant, must progressively become worse and lead to an early death.

She is evidently having pre-uremic symptoms. It is worth while stopping for a moment to notice how similar they may be to the symptoms of acute intestinal obstruction. Not infrequently when we see a patient with pain in the abdomen, with constipation, nausea and vomiting, the symptoms fit together into a picture of obstruction, whereas the patient may in fact be uremic. Even a high non-protein nitrogen does not help us, because in intestinal obstruction the non-protein nitrogen is high. With this story of headaches I think the diagnosis is not difficult.

"A third sound was audible." That makes a pretty full diastole. I wonder if one could pick up a diastolic murmur and a third sound too unless it were an aortic diastolic murmur. The murmur of mitral stenosis, if it were that, would be localized at a point just within the apex, not at the left border of the sternum. The mitral diastolic is a very localized murmur. We get it at just one place, and if we move the stethoscope half an inch it is not there. The characteristic murmur which one would expect at the left border of the sternum is the diastolic of early aortic regurgitation, and that we could not describe as a "low pitched" murmur. We should describe it as a high pitched blowing murmur, even though slight in intensity. So we have observations which do not allow us to decide whether this is mitral or aortic. The chances are that there is an aortic murmur. She has aortic dilatation. It might perfectly well happen that the ring would be dilated enough to get a little regurgitation. If that were the case, however, the diastolic pressure would come down, and it does not. The blood pressure is 270/160 to 215/125.

She has evidence now of albuminuric retinitis.

Now we have, superimposed on a kidney that is primarily at fault, evidence of chronic passive congestion. I think we may explain the red cells and granular casts in the urine as evidence of chronic passive congestion.

"One of three stool examinations showed a strongly positive guaiac," but the other two did not. One can have ulcerations of the gastro-intestinal tract during the pre-uremic and uremic stages. Either we have to assume that was the case here or else we must disregard this one strongly positive guaiac. I think it is wiser not to disregard it. Whether we can explain it or not I do not know.

You have heard me several times try to find a cerebral lesion in these cases of uremia with paralysis or coma or convulsions, and later the autopsy showed no cerebral hemorrhage. So it is quite possible to have all the signs of increasing intracranial pressure, — high spinal fluid pressure, enlargement of the retinal veins and so forth, and typical cerebral symptoms, coma, convulsions and paralysis,—without any gross cerebral hemorrhage shown at autopsy. I hope Dr. Mallory will say a word or two about that, because it was my feeling very definitely

until recently that by searching one would find hemorrhage in many cases of uremia.

The irregular breathing can be explained as Cheyne-Stokes breathing as the result of poisoning of the respiratory center by some products not excreted by the kidneys—what, we do not know.

Do the thick speech and loss of vision mean spasm of any one of the cerebral vessels?

We should like to be able to prophesy here whether they will find any actual gross pathology in the head. I do not think we have to hypothesize a cerebral hemorrhage. If she had a real hemiplegia then there would probably have been hemorrhage, but with uremia we can have as much as this without hemorrhage. I think we have here primarily a vascular nephritis resulting in tremendous hypertension, and all the other changes are secondary to that hypertension,—dilated and hypertrophied heart, dilated aorta and evidence of chronic passive congestion. Do we have to assume any other lesions in the heart? Are we likely to find an actual sclerosis involving the smaller vessels of the heart? I do not know. I think that a pretty normal heart working under such pressure as this did would come to the sort of grief this heart came to. I do not think we have to assume cardiosclerosis or bundle branch block. I am going to guess that very little will be found in the way of sclerosis in the heart, although I do not know whether we ever get marked sclerosis in the small vessels in the kidney without having sclerosis in the smaller vessels elsewhere in the body. Can we think of the kidney as being a rather localized point of sclerosis?

Dr. Tracy B. Mallory: No; I think we can always get changes in some at least of the other organs. I have never seen the spleen, for instance, uninvolved when the disease was at all advanced in the kidney. The distribution is very variable, but the heart is almost always excluded. Whereas the large vessels of the heart are among the early ones to be involved in atheroma, the arterioles are among the last to be involved in the hyalin degeneration associated with hypertension and vascular nephritis.

A Student: What about the white cells in the urine?

Dr. Fremont-Smith: I should say that was just a local infection, cystitis.

A Student: I should think they might be connected with nephritis.

Dr. Fremont-Smith: We should not get white cells in chronic nephritis. We get some white cells in subacute nephritis. Has this infection been working back into the pelvis of the kidney and causing pyelitis? I do not see any evidence for or against it.

A Student: What do you think was the terminal cause of death?

Dr. Fremont-Smith: Uremia, or else a vascular accident from this tremendous hypertension. She did not die a cardiac death; she died either a toxic death or a mechanical death from blood in the skull.

A Student: Is it not necessary to be pretty careful in making a diagnosis of essential hypertension in the presence of such urinary findings as those?

Dr. Fremont-Smith: I think there is a question as to whether there is any such thing as essential hypertension. We do get hypertension in older people who go quite a long time with nothing abnormal in the urine and no changes in the renal function tests, but at autopsy those people show changes in the kidneys of the same sort that we find here.

Dr. Mallory: Every one I have seen has so far.

Clinical Discussion

at staff conference

Dr. Howard B. Sprague: I saw her once at the first admission. I merely made a diagnosis of malignant hypertension, whatever that may be, and suggested that she see Dr. Palmer, who is especially interested in cases of this type.

Dr. Robert S. Palmer: I followed her in the Outpatient Department trying unsuccessfully to get her to follow a definite outline of palliative treatment. She did not take digitalis or anything else. She was a very difficult personality. She had had three illegitimate pregnancies and was a delinquent in the care of the State. There was a strong family history of vascular disease, three of her immediate family having had hypertension. She seemed to have definite malignant hypertension at twenty-four, and conformed to the type of person who is subject to it. Once or twice in the Outpatient Department she got considerable relief from headaches by venesection, but not much help for her paroxysmal dyspnea. I wondered if it would not have been a good plan to do a lumbar puncture when she was in the hospital, assuming that it might have been a hypertensive crisis and not uremia. Did she get any help at the times when she was bled?

Dr. Halsted: Yes, she did. She was bled twice with considerable relief.

Dr. J. H. Means: We have been discussing what type of nephritis this was, glomerular or not. I think it was not; I think she had arteriolar disease, malignant hypertension with a renal lesion which was entirely vascular. I think this case probably represents an entirely different disease from the other two cases, 18102* and 18132**.

Dr. Mallory: I think that is entirely true.

*Case 18102, published March 10, 1932.
**Case 18132, published March 31, 1932.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Malignant hypertension with cardiac failure.
Chronic nephritis.
Uremia.

DR. MAURICE FREMONT-SMITH'S DIAGNOSES

Vascular nephritis.
Malignant hypertension.
Hypertrophy and dilatation of the heart.
Dilatation of the aorta.
Chronic passive congestion.
Uremia.
Possibly a cerebral vascular accident.

ANATOMIC DIAGNOSES

Vascular nephritis, malignant type.
Arteriosclerosis.
Hypertrophy of the heart, hypertensive.
Chronic endocarditis of the mitral valve, rheumatic, without deformity.
Acute endocarditis of the mitral and aortic valves.
Bronchopneumonia.
Cholelithiasis.
Old operative wound: hysterectomy.
Cerebral infarcts.
Cortical atrophy.

Pathologic Discussion

DR. MALLORY: This case is the best example of malignant hypertension we have had in this department in the past five years; in fact it is histologically the only typical one, I should say.

The kidneys in such a case are always interesting. I was asked before the autopsy what we ought to find. I thought we should find a hypertrophied heart and that the kidneys would weigh between 250 and 150 grams; that is, they would be decreased in size, but probably not less than half the normal size, in contrast to the contracted kidneys of chronic glomerular nephritis, which frequently weigh less than 100 grams. I thought that when we stripped the capsules we should have a rough surface showing a combination of fine and coarse granulations, very red, in contrast to the grayish white surface with yellow speckles seen in chronic glomerular nephritis. I thought that the cortex would measure three to four millimeters against one or two which we should get in the end stage of glomerular nephritis.

At autopsy we found a pair of kidneys weighing 200 grams. The capsules left a red finely granular surface with occasional deeper pits, composed of grayish granules with a deep red background, giving a general effect of red kidneys. The cortex measured in two places 3 millimeters in thickness. Grossly and microscopically the kidneys would do for textbook pictures. There were very acute lesions in all the arterioles consisting occasionally of fibrinous thrombi, more often of hyalinization, and lipoid degeneration of intima and media. In the larger vessels there were marked intimal thickening and very marked elastica reduplication.

Similar changes were found elsewhere in the body in a distribution very characteristic of these cases. The lesions are usually more marked in the spleen than anywhere else; in fact they are so common in the spleen that one cannot make any guess from the appearance of the splenic arterioles whether or not there was hypertension in life, because nearly anybody over forty is apt to show changes there. The kidney usually comes next. Then we begin to find similar changes in the pancreas and in the capsule of the adrenal, and then quite often in very acute cases the intestinal tract becomes involved. We did not find any very definite lesions in the skin. If we had examined the retinae we should certainly have found something there.

These cases of vascular nephritis can be roughly divided into two fairly definite groups, one of which is usually styled benign and the other malignant. The benign type is very rarely seen in anyone much under fifty at the time of autopsy. The disease may start at forty, even a little earlier, but it does not kill people until ten or fifteen years after the onset, so that at autopsy they are apt to be in the late fifties and early sixties. We do not see it so often in people in the seventies, eighties and nineties because the victims die before they reach that period. In contrast, when arteriolar degeneration occurs in a person in the twenties or thirties, especially in a person as young as this, it runs a rapid malignant course of which the upper limit is about four years. Sometimes death occurs in three or even two years. Hypertension is usually the outstanding symptom. They run a long time without much evidence of renal insufficiency, and cardiac failure, secondary to hypertension, or cerebral hemorrhage may kill them when the renal function tests are still almost negative. In this case there was an extraordinarily good function, about 70 per cent. Often, however, they do eventually go into uremia. I think there is no doubt that this girl was uremic at the end. I also have a definite impression that these cases become uremic with a much lower nitrogen retention than in glomerulonephritis. In the latter the nitrogen is quite apt to be well over 100 before the patient becomes uremic. On the other hand these malignant vascular cases may begin to show uremic symptoms with a non-protein nitrogen not much over 50. At 60 and 70 symptoms frequently begin to appear. Her non-protein nitrogen was about 80 at the time she began to show slight twitchings. She did not have generalized convulsions or any marked signs of uremia.

Incidentally there was no cerebral hemorrhage.

DR. FREMONT-SMITH: Were there any ulcerations of the gastro-intestinal tract? Do they not rather frequently occur in uremia?

DR. MALLORY: Very frequently. Somewhat over 50 per cent of the cases do show a marked enteritis either in the large intestine or the terminal ileum. They may not go to ulceration, but they are more apt to be shown by a little thickening and little petechial hemorrhages. You would find however that it would be enough to give a positive guaiac.

A STUDENT: Was there any insufficiency of the aortic valve?

DR. MALLORY: She had a very definite rheumatic involvement of both the aortic and mitral valves without however any valve deformity. Our impression at autopsy was that there probably was a little mitral stenosis and we doubted if she had aortic insufficiency, but the lesions were so slight that it was difficult to be sure. There was no marked regurgitation, but she may have had a little. The aorta was not markedly dilated.

DR. FREMONT-SMITH: We cannot say then the arteriosclerosis in the retinal vessels runs parallel with the condition in the brain.

DR. MALLORY: I think it runs parallel to the condition in the kidney, but not necessarily to that in the rest of the brain. I am much more skeptical of that.

A STUDENT: How about the vessels we can palpate?

DR. MALLORY: They have no relation at all to hypertension I am sure. This girl had extreme hypertension for at least two years, yet the peripheral vessels showed no marked degree of sclerosis.

A STUDENT: In so-called essential hypertension do you get changes in the retinal vessels?

DR. MALLORY: Yes, I believe we do. I think this case is essential hypertension. Not everybody would agree with me as to that. Once in a while we see an apparently identical case recover, but they are very rare. I have recently seen such a case. A diagnosis of malignant hypertension was made eight years ago, when the patient was thirty-one years old. He was seen by nearly every important internist in Johns Hopkins, Chicago and Boston; they all agreed on the diagnosis, and he is better now than ever before.

DR. FREMONT-SMITH: He still has a slight hypertension, but it is getting definitely lower each year.

CASE 18352

EMPYEMA WHICH FAILED TO RESPOND TO TREATMENT

CHILDREN'S MEDICAL DEPARTMENT AND SURGICAL DEPARTMENT

PRESENTATION OF CASE

DR. RALPH W. DAFFINEE*: An Italian girl baby ten months old entered the hospital through the Outpatient Department because of persistent cough of four weeks' duration.

The birth was at term and was uncomplicated. The child was breast fed for two months, then weaned to a formula the composition of which is unknown. At entrance she was eating solid food. She had received orange juice but not cod liver oil. She had never been ill until the onset of the present illness.

The mother was in good health. The father was confined in a State institution at the time of the baby's illness. There were four older children, all in good health. The mother had had numerous miscarriages scattered between the normal pregnancies. There was no history of familial disease.

Four weeks before entry the baby caught cold and began to cough. After a week she seemed to be getting better, when she suddenly became very sick again. She was feverish, coughed a great deal and had difficulty with breathing. The mother thought she seemed blue. At times she coughed up yellowish material. As she seemed to be getting steadily worse, her mother brought her to the Outpatient Department. Her appetite had been failing during the illness. Her stools had been normal except for a green diarrhea lasting for one day shortly before entry.

On entry she looked extremely ill and toxic. She was poorly developed and had a waxy pallor. There was considerable respiratory difficulty, with an expiratory grunt. The cough was dry, hacking and unproductive.

At entry the temperature was 99.4°, the pulse 160, the respiration 66.

The child weighed only 14 pounds 5 ounces. There was bulging of both ear drums. The posterior cervical glands were slightly enlarged. There were no teeth. The right lung was clear to auscultation and percussion. The left base was flat and there was impairment at the apex. The breath sounds in the left back were nearly completely suppressed and in front were diminished but definitely tubular. The heart was displaced to the right; the right border of dullness was 4 centimeters from midsternum. No murmurs were heard. The abdomen was normal. There were no abnormal reflexes.

The urine, stools, Wassermann and tuberculin tests were negative. The red blood cell count was 3,660,000, hemoglobin 60 per cent,

*Recently resident on the Children's Medical service.

white cell count 23,450. The differential count showed 50 per cent polynuclear neutrophiles, lymphocytes 36 per cent, large mononuclears 3 per cent, myelocytes 6 per cent.

Course in the hospital. Shortly after the admission of the child the ear drums were incised with escape of considerable pus. A chest tap was done with removal of 20 cubic centimeters of thick yellow material from the left side of the chest. A smear showed Gram-positive diplococci. The child was transferred to the Surgical service for trocar thoracotomy. Half an ounce of thick creamy pus was drawn off at operation and an ounce every hour following operation as long as any remained in the pleural cavity. The temperature, pulse and respiration rose and the child appeared to be very ill.

On the fourth day following operation she was given a transfusion of 100 cubic centimeters of citrated blood. The chart of the temperature, pulse and respiration began to show a downward course but she still appeared very ill. The red blood cell count had fallen from 2,500,000 to 2,000,000. Little or no fluid could be aspirated from the chest.

On the eleventh day following operation she was given another transfusion, this time of 150 cubic centimeters. There seemed to be no marked change in the next few days. Her temperature remained at 102°, her pulse at 160 and her respirations above 50. Her white blood cell count began to fall. On the twenty-third day it had been 43,000, on the twenty-seventh day it was 18,000 and on the twenty-ninth day 9,000.

For the next two weeks her condition was about the same. She seemed much brighter at times, but the temperature never became normal. Slight infection developed about the transfusion wounds. The chest drained considerable pus. She was therefore placed on constant suction. Twenty-one days after operation she was given another transfusion of 150 cubic centimeters into the right ankle vein. The following day there was edema of the left foot and ankle. The temperature fell to about 100°, but in the next few days rose again; the curve swung between 104° and 100° until death. The edema of the ankle continued. She began to develop furuncles, and large glands appeared in the left groin. Thoracentesis was done to determine whether an encapsulated empyema was present, but no pus was found. On the thirty-sixth day after operation improvement was noted, but on the following day she was much worse, unusually apathetic, with marked pallor and dyspnea. A crop of pustules appeared on her back and buttocks. Transfusion of 100 cubic centimeters of citrated blood was done, but without improving her condition. Edema of the right eyelid appeared. On the fortieth day after operation all the edema had subsided, but she developed a necrotizing eruption over the scalp and over the lower back. She went steadily downhill during the next two days and died forty-two days after operation.

Clinical Discussion

Dr. Robert R. Linton: I was interested in this child, as I was responsible for her surgical care.

In the first place, regarding the question of diagnosis when the child entered. We had a history that four weeks before entry she caught cold and began to cough. This lasted for a week and seemed to be getting better, when she suddenly became very ill again.

Empyema usually starts in one of three ways. (1) It may start very acutely as the primary disease, or (2) it may follow pneumonia but blend in with the pneumonia so that it is a little difficult to tell just when it starts, or (3) it may follow the crisis, coming on gradually a few days or a week after the temperature has returned to normal. I think this happened in this case, although I cannot be sure as I did not observe the child at the time. She was ill for a week and she apparently recovered by crisis. Following the crisis she started to run a little temperature which finally went up to the degree it was when she entered the hospital. I think she probably had had empyema for three weeks; it was present for two and a half weeks at least.

On going over the patient all the physical signs for empyema were quite definite. The classical signs are dullness to flatness over the chest, diminished breath sounds and displacement of the heart and mediastinum. This patient had all three.

The question comes up as to treatment. I am pretty well convinced that once one has made a diagnosis of empyema—and that is done only by doing a chest tap as a final diagnostic measure—one should drain the pus in one of several manners. The choice depends a good deal on the type of pus which is found. If it is pneumococcus pus I think it is best to operate, giving either closed or open drainage. If the empyema is of streptococcic origin one should not be hasty about draining off the pus. The reason for this difference in treatment is that in pneumococcus empyema there are a great many adhesions which surround the empyema and the lung is usually very much bound down in the thorax, so that if air does get in during operation one does not produce generalized pneumothorax or disseminate the pus as one is likely to in doing an open operation on a streptococcus empyema. We make the diagnosis of the kind of pus by making a smear of the pus as soon as we do the chest tap. In a matter of only three or four minutes we are usually able to decide whether it is pneumococcus or streptococcus. One may see a case of staphylococcus, but that is very rare. The character of the pus also aids in the differentiation, as pneumococcus pus is thick

and creamy while staphylococcus pus is thin and watery.

In this case we smeared the pus and found that it contained numerous Gram-positive diplococci, probably pneumococci. The pus was thick and creamy in consistency. The rest of the examination of the child seemed to be essentially negative at that time except for her ears. A trocar thoracotomy was done under novocain anesthesia. In this case I think we got into the ninth interspace, perhaps one lower than we should have liked to be in. We got pus, however, and drew off at least half an ounce at that time. One does not expect a great deal of pus in a baby only ten months old. I do not believe she had more than 4 or 5 ounces at the most.

The patient did not do very well after the trocar thoracotomy. She was transfused several times. Dr. Daffinee told you that she was put on constant suction. That is an addition to our treatment of empyema instituted during the past two or three years. It is very gentle suction. We begin with a few centimeters of water pressure and gradually increase it. The advantage of using constant suction is that it tends to decrease the empyema cavity by causing the lungs to expand a little more rapidly. The cavity continued to hold one ounce to one and a half ounces of saline even up to death. Another striking thing is the differential count. She had only 54 per cent polynuclears, which is rather a low percentage I think for an acute infection by pneumococcus, and she had a white blood count of 22,000, all of which indicates that she had a very low power of resistance.

We had great difficulty in giving her transfusions because she had such very small veins. We usually succeed in getting in 100 cubic centimeters. Once we succeeded in getting in 150 cubic centimeters, which is a fairly large amount for such a small child.

Another point of importance in this record is that she developed left inguinal adenitis and edema of the left foot and ankle three or four weeks after she came in. I examined her carefully at that time. Dr. Higgins and several others also saw her. We were unable to explain the cause of the swelling of the foot and leg and the inguinal adenitis. At that time there was some question whether she might not have meningitis. She had lain with her neck in hyperextension for several days. Repeated examinations for Kernig however were negative, so that we did not think she had meningitis and a lumbar puncture was not done. I mention that because I wish you to realize that we did examine her legs. We put her through a great many motions. Ordinarily I think we should have picked up something of what the pathologists found at autopsy if the patient had not been so ill.

The later course was merely gradual failure I should say. Strange to say, the swelling of the legs disappeared entirely and the adenitis to some degree. I think that during the last week we probably neglected her legs because of their return to normal size.

We thought her death was due to an overwhelming pneumococcus infection which eventually led to a septicemia.

These x-rays are of interest. This film, taken on the day of admission, shows complete obliteration of all the lung markings on that side. There is displacement of the heart and mediastinum to the right. The trachea is considerably displaced. This next film shows the position of the catheter. It looks as if the tube were beneath the diaphragm. This is due to marked gastric distention, pushing the diaphragm up and the tube is down in the costophrenic angle.

Here is a film which shows the tube well above the diaphragm. The next one taken five days after the trocar thoracotomy shows most of the fluid removed. There is considerable consolidation in the lower lobe.

In the next film the chest does not look so well. There is considerable air in the pleural cavity which shows by taking the film with the patient in the lateral position. There is essentially not much change in the ones taken during the last two or three weeks.

Dr. Harold L. Higgins: When we saw the patient at entrance we felt quite certain there was empyema. After establishing the diagnosis we referred the case to the surgeons, but we kept in touch with her while she was on their service. With the failure to improve under treatment we felt that there was active pneumonia in addition to the empyema. In empyema cases we see those who have pneumonia and those who do not seem to have it. Patients with empyema without pneumonia are usually breathing quite quietly. They may be having a temperature but they ordinarily are fairly comfortable, at least as far as respiration is concerned, provided there is sufficient drainage. In other cases we find the patients breathing rapidly and having the clinical symptoms of pneumonia in addition to empyema. As we examined this child in the last two weeks of her life we felt very definitely that in addition to empyema she must have some bronchopneumonia which was contributing to her condition. When a patient with empyema has pneumonia also, one should be much more slow in removing fluid and more careful to avoid a pneumothorax than when a patient has merely a purulent exudate with no clinical symptoms of active pneumonia.

There is also a point in regard to the probability that she had had empyema two weeks or more when she entered. I have seen an empyema with fluid filling the chest develop in

twenty-four hours. In this case the empyema may have been present for two weeks, but it is difficult to say from this history exactly how long the duration was.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Empyema.

ANATOMIC DIAGNOSES

Septicemia, pneumococcus type V.
Empyema, left pleural cavity.
Atelectasis, bronchopneumonia, chronic bronchitis and small abscess of the left lung.
Operative wound: thoracotomy.
Septic arthritis of the hip, bilateral.
Psoas abscess, bilateral.
Congenital anomaly, failure of fixation of the cecum and the ascending colon.
Accessory spleen.

Pathologic Discussion

Dr. John I. Bradley: Starting the postmortem examination with the thoracic cavity, we noted first a thoracotomy wound in the left lower lateral thoracic wall in about the midaxillary line. This led into a large empyema cavity which occupied the major portion of the thoracic cavity, more particularly the lateral and the lower posterior portions. The lung was collapsed to less than half the normal size and displaced into the upper and medial portions of the thoracic cavity. The pleura was quite firmly adherent over the collapsed lung. The large empyema cavity was covered by a shaggy fibrinous coating. The open thoracotomy provided adequate drainage and there was practically no fluid in the cavity. The collapsed lung on section was homogeneous reddish-gray in color, with no areas of consolidation. It was apparently a compression atelectasis of the lung. There was one small cavity filled with purulent fluid which lay just beneath the pleural surface in the upper portion of the lower lobe.

When the rather lengthy examination of the thoracic cavity was concluded most of the audience dispersed and a rather hurried completion of the routine postmortem examination was begun. On starting to free the bladder there was a sudden gush of a large amount of thick greenish pus. Inspection showed a large fluctuant mass, pressure on which caused a copious outpour of pus. On opening the abscess we found that it overlay the psoas muscle on that side. The cavity ended very definitely at its upper end. The upper portion of the psoas muscle was negative. The spinal column was negative. There was no connection with the empyema cavity. On following the abscess down we found that it passed into the upper thigh, that it surrounded the head and neck of the femur anteriorly, laterally and medially, that it communicated with the joint cavity and that the hip joint was filled with the same thick purulent fluid. The head of the femur on the left side was small, irregular and eroded and was rather freely mobile at the epiphyseal line. The surface was infected. Turning to the other side we found a rounded fluctuant mass, considerably smaller, in the right iliac fossa. We were able to trace that down into the thigh and found that it communicated with the hip joint on that side.

The bacteriologic reports on the cultures were pneumococcus in the heart's blood and in the pus from the right hip joint, staphylococcus in the pus from the empyema cavity and hemolytic streptococcus in the small abscess in the left lung.

The examination was otherwise essentially negative.

A Physician: How do you explain that abscess formation?

Dr. Bradley: I think its origin was in a septic arthritis which had extended upward. I should say it was a metastatic process.

Dr. Higgins: Was there no abscess anywhere else?

Dr. Bradley: Only the small abscess of the left lung.

Dr. Linton: I think it is surprising we did not notice that the child was very tender when we moved that leg. Apparently she was not. We could move it and twist it without apparent discomfort. When we did a Kernig test the child did not cry any more than when we did anything else to her. I think that was probably one reason why we missed it.

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THE B. & M. CASE

PHYSICIANS interested in the suppression of alleged unapproved remedies should read the account of the suit in the Federal District Court of Baltimore against the F. E. Rollins Company which put B. & M. on the market.

Some interesting facts brought out in this suit may engage the attention of Massachusetts people. The defense was conducted chiefly by Melvin M. Johnson, a Massachusetts lawyer, who in addition to acting as counsel appears to have been the president of the Rollins Company.

An interesting feature of the testimony, introduced by the defense, is that which was submitted by Dr. David L. Martin of Boston. If this David L. Martin is the David Lorenzo Martin, a graduate of Boston University School of Medicine, as appears in the directory of the American Medical Association, and also a Fellow of the Massachusetts Medical Society, as set forth in the directory of the Officers and Fellows of the Massachusetts Medical Society, some concern will be felt in Boston medical circles.

In the report of the proceedings in the *Journal of the American Medical Association* it appears that Dr. David L. Martin is a graduate of a homeopathic school and prior to that he had been a preacher for nine years. Inquiry of the Boston University School of Medicine yielded the information that David Lorenzo Martin is a graduate of that school and that he is the holder of the degree of Bachelor of Sacred Theology which was one of his premedical acquisitions. Thus far circumstantial evidence would seem to indicate the desirability of ascertaining whether David L. Martin, the recorded endorser of B. & M., is the David Lorenzo Martin who is a graduate of Boston University School of Medicine, a clergyman and a Fellow of the Massachusetts Medical Society. The statement that Dr. David Lorenzo Martin is a clergyman is based on reports furnished by the Dean's office of the Boston University School of Medicine to the effect that he still preaches occasionally, thus combining theology with medicine.

If the report of the testimony of David L. Martin cannot be attributed to David Lorenzo Martin, much relief will follow among those who deal with irregularities of behavior among doctors. If David L. and David Lorenzo are one and the same person, a certain responsibility seems to lie with the medical profession, for David L. evidently tried to bolster up a concern, the product and claims of which led to a Federal adverse decision.

Dr. David L. Martin is reported in the *Journal of the American Medical Association* to have testified "that B. & M. was such a wonderful preparation that, when applied, the character of the eruption that it invoked indicated the nature of the ailment for which the patient was being treated." He then went on to describe the eruption characteristic of "flu" or "a mixed infection" and several other claims which indicate astounding ignorance or crude dishonesty.

The relation of Dr. Pease, as set forth in the record, to the Rollins Company is of little interest to the medical profession outside of New York State except as it applies to the history of this company but the record of Mr. Johnson, counsel and president, who is a Massachusetts lawyer, is open to question. If the grievance committee of the Massachusetts Bar Association does not shown concern about Mr. Johnson's relations with this company, it may be because it has too many irregularities on the part of other lawyers before it which may prevent its going so far afield.

Whether any action respecting Dr. David L. Martin's association with the Rollins Company and his deplorable ignorance of sound medical practice is advisable will depend on the question of the identity of the David L. and David

Lorenzo because throughout the testimony of David L. Martin, as reported in the *Journal of the American Medical Association*, there is abundant evidence tending to indicate that he is not qualified to practice medicine because in treating "blood poison" he found that the use of B. & M. had never failed.

The *Journal of the American Medical Association* is entitled to the gratitude of the public for its account of this trial. The cases of Dr. Martin and Mr. Johnson, however, should not in our opinion be regarded as closed.

REMEMBER THE DEACONESS HOSPITAL

THE Superintendent of the Deaconess Hospital in Boston has been quoted in substance as announcing that for the first six months of the current year, by reason of drastic economies, this institution has managed to carry on without a deficit.

An ominous prediction, however, appears in a statement to the effect that it will be impossible to continue free and part pay service in the same measure as in the past without an accession of new funds.

There are several very important features of the work of the Deaconess recognized throughout the country with respect to cancer, diabetes and thyroid disorders.

The *Boston Post** publishes a letter which explains the situation:

$65,000 REQUIRED

"The debt of the Deaconess Hospital must not be increased one cent either by capital expenditure or operating deficit. The Baker Unit will not be built until $50,000 additional are available to cover the entire cost of the building and $15,000 yearly for 20 years are guaranteed for maintenance. At this writing, approximately one-fourth of this requirement above Mr. Baker's gift of $250,000 is assured. The balance should be raised before Sept. 1, when some of the gifts lapse.

"The time allowed is not too short, because, thanks to the coöperation of the press, all who are likely to be donors are informed and the total to be raised is so small. Indeed only $65,000 are required within the next six months—$50,000 to complete the price for the contract and $15,000 as the first of the 20 yearly payments for maintenance. All gifts are solicited with the understanding that the entire sum required is subscribed and that the building begins immediately.

"By a vote of the executive committee of the Deaconess the guarantees for future payment must be acceptable to the finance committee, upon which are the treasurer, Mr. W. Jesse Fowler, a vice-president of the First National Bank, and Mr. Wilmot Evans, president of the Boston Five Cents Savings Bank.

"Special gifts, as memorials, could be made for the laboratory for research in chronic disease or for the unique children's department. The research laboratory occupies one entire floor. The cost of this and of the children's department makes most of the difference between Mr. Baker's original subscription and the contractor's bid.

*August 26.

"The Baker clinic makes possible economic advantages for the Deaconess Hospital which are almost vital. It will be a productive investment, thus meeting the requirements of any welfare board, a fresh addition to Boston's and New England's resources for health, and an aggressive attack upon chronic diseases, particularly diabetes and cancer, which are steadily increasing as we cease to die young."

The *Boston Herald** gives a description of the penthouse as follows:

"A hospital equipped with a penthouse, a gymnasium, an outdoor playroom for children, and rooms arranged so that a member of the family may reside with the patient to acquire knowledge of home care, are only a few of the features of the new 47-bed hospital to be erected by the New England Deaconess Hospital this fall. The building, which will contain five stories, will be of red brick with limestone base and trim, and will face the Deaconess on the Riverway. It will contain the most modern of hospital equipment, and the rooms will be so constructed that the patient occupancy can be doubled if necessary.

"A unique feature of the building will be two four-bed children's wards, one for boys and one for girls, suffering with chronic diseases. Between these two wards will be a small dining alcove where they will obtain their meals, and be instructed regarding diet. Leading directly from them will be a stairway to a penthouse on the roof in which will be a gymnasium for play and exercise."

"The Baker clinic will provide amusement and exercise for the patient while there, and instruction regarding future care after being discharged."

An interesting feature of the work of this hospital is the sacrificing devotion of doctors, nurses and all of the employees in maintaining the greatest possible efficiency in dealing with the ills of its patients.

Any crippling of the hospital functions must be obviated, but this means more generous contributions. Every little helps and concerted action by doctors and the friends of patients will bring the required support.

*August 25.

THE HEALTH OF THE RETURNING VACATIONIST

STATISTICS show that during the summer months mortality rates drop to the lowest averages for the year and that there is apt to be a slight rise in September to be followed by a more or less interrupted increase extending to and through the winter months.

Two factors have in some years seemed to contribute to the September rise; first, the condition of the vacationist on returning home, and, secondly, the increase among school children of communicable diseases.

Some people have had to acknowledge that vacations have not brought about the hoped-for accession of vigor, but on the other hand the intrusion of a pointed question is timely as to whether the unwonted activities and changes in habits have not done more harm than good in some instances, and that some children may be

carriers even if not actively ill, and start a blaze of infections among fellow students.

May it not be appropriate for physicians to suggest that the advocated health survey should be made following the usual vacation period and especially with children before taking up school duties.

Doctors are not, on the average, busy at this season and it may be a good time to advance the wisdom of health examinations. Both the physician and his clientele would benefit thereby.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

TRUESDALE, P. E. M.D. Harvard University Medical School 1898. F.A.C.S. Surgeon-in-Chief, Truesdale Hospital, Fall River. His subject is "Gastric Ulcer Associated with Diaphragmatic Hernia." Page 385. Address: 151 Rock Street, Fall River, Mass.

CORVESE, ANTHONY. M.D. Tufts College Medical School 1912. F.A.C.S. Assistant Surgeon, Rhode Island Hospital. Gynecologist, Charles V. Chapin Hospital, Providence, R. I. His subject is "Acute Perforation of Peptic Ulcer." Page 390. Address: 485 Broadway, Providence, R. I.

KENNEDY, ROBERT H. B.A., M.D. Columbia University College of Physicians and Surgeons 1912. F.A.C.S. Attending Surgeon, Beekman Street and Stuyvesant Square Hospitals, New York City. His subject is "Emergency Treatment of Extremity Fractures." Page 393. Address: 115 East 61st Street, New York, N. Y.

MOORE, GEORGE A. S.B., M.D. Harvard University Medical School 1911. F.A.C.S. Surgeon, Moore Hospital, Brockton, Massachusetts. Consulting Surgeon, St. Luke's Hospital, Middleboro, Massachusetts and Bridgewater State Hospital. His subject is "Intussusception in Adults." Page 395. Address: 167 Newbury Street, Brockton, Mass.

WHITE, WILLIAM A. JR. S.B., M.D. Harvard University Medical School 1921. F.A.C.S. Junior Visiting Surgeon, Boston City Hospital. Assistant Surgeon, Massachusetts Women's Hospital. Assistant in Surgery, Harvard Medical School. Address: 270 Commonwealth Avenue, Boston, Mass. Associated with him is

COONEY, EDWARD A. Ph.B., M.D. Harvard University Medical School 1926. Assistant to Visiting Surgeons, Boston City Hospital. Assistant in Surgery, Harvard Medical School. Address: 270 Commonwealth Avenue, Boston. Their subject is "A Nonoperative Treatment of Carbuncles." Page 398.

MEIGS, JOE V. A.B., M.D. Harvard University Medical School 1919. F.A.C.S. Surgeon to Out-Patients, Massachusetts General and Collis P. Huntington Memorial Hospitals. Assistant Surgeon, Palmer Memorial Hospital. Surgeon-in-Chief, Vincent Memorial Hospital, Boston. Surgeon, Pondville Hospital, Norfolk. His subject is "Trichomonas Vaginalis Vaginitis." Page 403. Address: 264 Beacon Street, Boston.

WOODMAN, J. B. A.B., M.D. Dartmouth Medical School 1903. F.A.C.S. Surgical Staff, Franklin (N. H.) Hospital. His subject is "Appendicitis." Page 406. Address: 336 Central Street, Franklin, N. H.

FITCH, EMERY M. M.D. Dartmouth Medical School 1905. F.A.C.S. Surgeon, Claremont General Hospital. His subject is "Inversion of the Uterus." Page 413. Address: Claremont, New Hampshire.

AN IMPENDING COMPLICATION AFFECTING MEDICAL PRACTICE*

COMMITTEE ON STATE AND NATIONAL LEGISLATION

8 The Fenway, Boston.

Dear Sir:

The Massachusetts Medical Society, founded in 1781 and now numbering about 4800 members, is confronted with an important problem and asks for your coöperation.

The chiropractors, a medical cult who call themselves "Drugless Healers", have obtained the requisite number of signatures of legalized voters and their Initiative Petition will appear upon the November ballot. Chiropractic is defined in said petition as "the science or practice of locating and adjusting by hand the malpositions of the articulations of the human spine". For the past ten years all of their bills have been defeated by legislative action and in 1932 they were given leave to withdraw by a vote of 151 to 50 in the House and 21 to 13 in the Senate.

The present law provides a single standard for all who practice the healing art in this Commonwealth and is satisfactory to all graduates of recognized medical schools including those from homeopathic and osteopathic schools.

The chiropractors ask for special privileges although our form of government abhors class legislation. Chief Justice Rugg of the Supreme Judicial Court has rendered an opinion in the case of Commonwealth vs. Zimmerman that a chiropractor practiced medicine without being lawfully authorized.

After an individual has successfully passed the examination of the Board of Registration in Medicine, he should be permitted to practice the method for which he is best fitted, be it allopathic, homeopathic, osteopathic or chiropractic. The sick, injured and infirm must be protected from those who are not able to comply with the present law.

*Copy of a letter sent to certain groups by the Committee on State and National Legislation of the Massachusetts Medical Society.

An Editorial in the *Boston Herald* of June 30, 1932 said in part, "We should not lower the bars for the benefit of those who cannot meet our reasonable requirements. New Jersey did some years ago, and in one year *536 Chiropractors* were licensed. The next year New Jersey put the bars up again."

We feel that this problem is a mutual one and that your organization should become active in opposing this petition. We would like to present more information at a personal conference if you are interested.

Very truly yours,

H. G. Stetson, *Chairman,*
T. J. O'Brien,
F. E. Jones,
A. W. Marsh,
Shields Warren, *Secretary-Treasurer.*

MISCELLANY

A RADIO MESSAGE PREPARED AND SPONSORED BY THE COMMITTEE ON PUBLIC EDUCATION OF THE MASSACHUSETTS MEDICAL SOCIETY FOR THE DEPARTMENT OF PUBLIC HEALTH

Medical Care*

BY ROBERT B. OSGOOD, M.D.

Most of you who are listening in have heard of the White House Conference called by President Hoover and organized by the Secretary of the Interior, Dr. Ray Lyman Wilbur, in 1929. This conference was directed by the President to consider and conserve the health and well-being of the children of the United States. It covered three years of labor by thousands of people. One of the pledges in the great "Children's Charter" which was drawn up reads as follows: "For every child from birth through adolescence, promotion of health, including health instruction and a health program, wholesome physical and mental recreation, with teachers and leaders adequately trained."

One of the main committees of this huge conference which considered every phase of child life was the Committee on Medical Care for Children. This committee attempted to find out three things: First, what is being given in the way of medical care; secondly, how adequate or inadequate the present medical care is; and thirdly, what ought to be done to fulfill the pledge in the "Children's Charter" which I have just read to you.

We have been asked to acquaint you very briefly with the findings of this Committee on Medical Care for Children. We shall tell you of the good things and the bad things the committee found, and some of the things which the committee thinks ought to be done to give the American child a better chance to be well and a better chance to grow up into a healthy-minded and healthy-bodied citizen of the United States.

No one can teach either the children or their parents how to be healthy unless he, himself, knows how to be healthy. So, the first task of the committee was to find out how well the various health agents, the physicians, the nurses, the social service workers and the dietitians who have to do with the feeding of children are being educated to give instruction in medical care.

The committee found that in many of the best medical schools too little time was being given to teaching the prevention and treatment of the diseases of children and that there were too few good courses offered which practising doctors could take when they found they needed "brushing up" in the newer methods of prevention and care. This department of medicine is called Pediatrics. The committee also found that much too little attention was being paid by most schools of nursing to instruction in how to keep children well, and how to nurse them when they were sick. It found that the supply of nurses specially trained to take care of children is very inadequate.

Medical Social Service, which was started over twenty-five years ago by Dr. Richard Cabot at the Massachusetts General Hospital, has proved of greatest value in the care of children in finding out the medical needs among the poorer families and arranging to have these needs met. The committee found that the supply of specially trained social workers of this sort was even more inadequate in most parts of the country than the supply of specially trained children's nurses.

It found that the so-called "dietitians" or child-feeding specialists were generally very well educated at that time, but that there were too few of them being educated.

The next task of the committee was to find whether the present facilities for the care of children in hospitals throughout the country were satisfactory. We are glad to be able to say that there are almost enough hospital beds assigned to children throughout the country to meet the needs, but sometimes the doctors who are obliged to take care of the children have had less training and experience in Pediatrics in many hospitals than is desirable for the best care of the children placed under their charge. This defect is being quickly remedied. There is great need, however, for two kinds of hospital beds for children. One is more widely distributed beds in the smaller communities for children sick with contagious diseases. People are learning that better care can be given to children who have such diseases as scarlet fever and diphtheria and measles in special isolation hospitals than in their homes. Most children seem quite as happy in a well-run sunny hospital as at home, and in all these diseases many complications may arise which may be anticipated and prevented better in hospitals than in homes, especially in small homes with large families, where there is no opportunity to have trained nursing.

The other need is for more convalescent homes

*Broadcast: May 24, 1932. Station: WBZ—Hotel Bradford. Time: 4:20 P.M.

for poor children who are suffering from the chronic diseases such as tuberculosis and heart disease, infantile paralysis, rheumatism and many bone and joint diseases. These children must now be sent home too early from the general hospitals in order to make places for those more acutely ill and also because they cannot pay the price for board which is necessary for those hospitals accepting only acute cases. Much treatment is, therefore, wasted, but would not be wasted if these children could fully recover in the fresh air and sunshine of a convalescent home out of the city dust and smoke. Such convalescent homes are much less expensive to run than the busy, fully-equipped city hospitals.

We shall now try to tell you of a few of the weak spots in our present national defences which should be made strong if the proper medical care of children is to be given.

Less than one-quarter of the children of the United States get the best work out of their body machines. The other three-quarters stand and sit, play and work, and even rest in such postures and positions that their muscles and their internal organs must work with unnecessary handicap and with unnecessary fatigue. In only a few widely scattered sections of the country is any general attention being paid to proper body mechanics, but in these sections the rewards of this attention are great in the way of better health and greater alertness and a greater sense of well-being. If effort along these lines can be made nation-wide and more well-run clinics in posture and body mechanics can be established we may expect in the next generation to improve materially the physical fitness of the race. The universal draft in the World War showed how disgracefully we need this as a nation.

There is great need also for more clinics for little children of preschool age who are deaf. There are said to be at present three million deaf school children in the United States and the problem of their serious handicap can be solved only by recognizing their danger before the school age and applying preventive and curative treatment early.

Now comes the startling finding of the committee. With the help of the existing social and medical agencies in different sections of the country, a survey was made of nearly 175,000 city living and country living children as to whether the now available well-established and proved preventive health measures were being used by parents before the children were really sick. The results of this nation-wide survey are alarming. Vaccination against smallpox is about the most certain preventive measure we have. Yet less than one-quarter of the city children throughout the nation have been vaccinated before they go to school and only about 1/15 of the children who live in the country. There are 55,000 unnecessary cases of smallpox in the United States each year. The danger has been recently emphasized in Fitchburg, Massachusetts, and its complete control proved possible by vaccination. Only about one-fifth of our nation's children have been given protective inoculation against diphtheria, yet in 1928 there were 5,000 seemingly unnecessary deaths from diphtheria in little children under five years of age.

Less than one-seventh of our young children have had their teeth examined and treated and yet nearly ninety-five per cent of school children show evidence of decay in their teeth which might have been almost entirely prevented by so simple a treatment as giving them the right kind of food to eat.

As the Chairman of the Committee on Medical Care for Children said in relation to the limited use that is being made of preventive measures, "Here is a challenge which every community is able to accept and with the surety of surprising results." The check-up in Massachusetts shows that we need very badly to accept this challenge.

A tremendous amount of information has been gathered by the thousands of experts who have been working without pay for the White House Conference. Attempts are being made to broadcast this information in many ways. This talk is a small attempt of this sort. Unless we use this information and make strong efforts to stop the wastage that is now going on, and to close up the breaches in the defences of child health, much of this unselfish labor which has been given so freely will have been useless. President Hoover's splendid ideal which inspired the Children's Charter will not be realized. We can help him realize it if we will. Never before has the American public been offered such a chance to rear healthy and happy and efficient children. The nation needs them. The information is being assembled and published. You must demand it and then not only put it into practice yourselves, but persuade others to do likewise. This is our clear duty if we love our children and our nation.

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR JULY, 1932

Scarlet fever, which has been particularly high for several months, is dropping off toward more normal levels.

Measles, which began to show an increase late in the season, is still somewhat above the usual number reported for this month.

Mumps and chicken pox are likewise slightly above normal levels.

Anterior poliomyelitis continues its low level begun earlier in the year, the number of cases reported for July being the lowest ever recorded except 1929.

The number of cases of diphtheria and typhoid fever reported continue to show a downward trend.

The prevalence of epidemic cerebrospinal meningitis, German measles and influenza is at a low ebb for this season of the year.

Whooping cough, lobar pneumonia and tuberculo-

sis, both pulmonary and other forms, were reported about the number of times expected for this month.

RARE DISEASES

Anterior poliomyelitis was reported from Lawrence, 1; Leominster, 1; Lowell, 1; Marblehead, 1; Marion, 1; Newton, 1; Total, 6.

Pellagra was reported from Acton, 1; Boston, 2; Total, 3.

Septic sore throat was reported from Amesbury, 3; Andover, 1; Ashby, 1; Boston, 7; Cambridge, 1; Dartmouth, 1; Greenfield, 1; Lowell, 1; Medford, 1; New Bedford, 1; Newton, 1; Somerville, 1; Total, 20.

MONTHLY REPORT OF CERTAIN COMMUNICABLE DISEASES

	Cases in Entire Pop.				Case Rates per 100,000 Pop.		
	July 1932	July 1931	Proso. Index*	Epi. Index†	July 1932	July 1931	Expected Rates‡
All Causes	5926	5771	—	—	137.9	135.0	—
Ant. Polio.	6	59	33	.2	.1	1.4	.8
Diphtheria	140	165	232	.6	3.2	3.9	5.4
Ep. C. S. Men.	2	6	5	.4	.02	.1	.1
Measles	1393	964	1290	1.1	32.4	22.5	30.0
Pneu. Lobar	101	92	99	1.0	2.3	2.2	2.3
Sc. Fever	602	482	328	1.8	14.0	11.3	7.6
Tbc. Pulmonary	434	429	395	1.1	10.1	10.3	9.2
Typhoid Fever	20	34	25	.8	.5	.8	.6
Whooping Cough	582	520	772	.7	13.5	12.2	18.0
Chicken Pox	441	370	—	—	10.2	8.7	—
German Measles	23	71	—	—	.5	1.7	—
Gonorrhea	605	632	—	—	14.1	14.8	—
Influenza	4	9	—	—	.1	.2	—
Mumps	341	270	—	—	7.9	6.3	—
Syphilis	349	337	—	—	8.1	7.9	—
Tbc. Other Forms	55	55	—	—	1.3	1.3	—

*This index is an attempt to estimate the number of cases based on the trend during the past years which can be expected to occur, and is for the purpose of comparison with the number of cases which actually did occur.

†This ratio expresses how prevalent the disease is compared with the index mentioned above; 1.0 indicates that the actual number of cases equals the expected number. A larger number means a greater prevalence, and a smaller number a lesser prevalence than expected. Thus 2.0 would indicate twice the expected number, and .5 half the expected number of cases. The method used to determine the indices is described in the August 18, 1927 issue of the *Boston Medical and Surgical Journal*.

‡Calculated from the Prosodemic Index.

Dysentery was reported from Chelsea, 1.

Encephalitis lethargica was reported from Chicopee, 1; Northampton, 3; Salem, 1; Total, 5.

Epidemic cerebrospinal meningitis was reported from Chicopee, 1; Leominster, 1; Total, 2.

Malaria was reported from Chelsea, 4; Easton, 1; Total, 5.

Tetanus was reported from Cambridge, 1; Chicopee, 1; Fall River, 1; Sharon, 1; Worcester, 1; Total, 5.

Trachoma was reported from Boston, 3; Worcester, 1; Total, 4.

Trichinosis was reported from Newton, 1.

THE SEVENTH HEALTH UNIT FOR BOSTON, MASSACHUSETTS

As illustrations of modern preventive medicine the several Health Units of the City of Boston, erected and equipped under the provisions of the George Robert White Fund, stand among the effective bulwarks against the invasion of disease.

The seventh in this group is now being erected on Whittier Street in the Roxbury district and completes the series for which plans were made about ten years ago.

The laying of the corner-stone of this structure was the occasion of a large open air meeting August 26, 1932, in front of the building in response to invitations issued by the Trustees of the George Robert White Fund.

The program was as follows:

Presiding, Mr. Edward L. Logan, Manager, George Robert White Fund.

Invocation, Reverend William J. Casey, Pastor, St. Patrick's Church.

Address, Dr. Charles F. Wilinsky, Deputy Health Commissioner, City of Boston.

Address, Mr. Richard D. Gleason, City Councillor.

Address, His Honor the Mayor, James M. Curley.

Benediction, Reverend David S. Klugh, D.D., Pastor, Peoples Baptist Church, Roxbury.

After the welcoming address by Mr. Logan and the invocation by Father Casey, Dr. Wilinsky gave an interesting account of the activities of the Boston Department of Health with an explanation of its relation to the several health units created under the terms of the bequest. He told of the importance of providing instruction to parents respecting the problems of maternity and the health of children.

A merited tribute to His Honor, Mayor Curley, for the influence in the inauguration and continued support given by the Mayor was a feature of this address.

Mayor Curley in his address gave an account of his association with Dr. Wilinsky in the creation and development of an experimental health unit which inspired the purpose of conferring with the late George Robert White. At successive interviews with Mr. White he gave detailed illustrations of the remarkable work of Dr. Wilinsky in the institutions which he, Dr. Wilinsky, had established and which evidently led to the clause in Mr. White's will which made possible these seven units.

He explained that the Fund has provided about two and a half millions for these seven institutions which are now under the control of the Boston Health Department and for which the city appropriates thirty-five thousand dollars annually. One especially unique feature in the will was emphasized which showed confidence in public officials in that the Trustees include the Mayor of Boston, Chairman Ex-Officio; the President of the City Council, the City Auditor, the President of the Chamber of Commerce and the President of the Bar Association, and he felt that this group would always assure the citizens of Boston of an administration devoted to promoting the health of the community efficiently and economically. At the close of the address the Mayor divested himself of his coat and in laying the corner-stone of the structure buried a copper casket which contained a copy of the appropriate clause of the will of George Robert White and the names of the Trustees.

The residents of that section of the City were present in large numbers with their children. The dignity of the occasion and the interest shown by the large attendance gave promise of a great and important addition to the health activities of Boston and a continuation of a service which has enabled the City to hold its place as one of the foremost municipalities of this Country in the application of preventive medicine.

CORRESPONDENCE

THE CASE OF DAVID LORENZO MARTIN

August 27, 1932.

Noel G. Monroe, M.D.,
39 Boylston Street, Boston.

Dear Dr. Monroe,

Referring to your letter in the *New England Journal of Medicine*, issue of August 25, 1932, page 383, the chairman of the standing Committee on Ethics and Discipline of the Massachusetts Medical Society bids me write and explain the situation of the case against our Fellow, David Lorenzo Martin of 419 Boylston Street, Boston. Charges that he was physician to the F. E. Rollins Company, Boston, the manufacturers of the B. & M. External Remedy, and was accustomed to prescribe that escharotic liniment in cases of pulmonary tuberculosis, were lodged with the committee in January, 1931. Shortly after, the chief of the Division of Food and Drugs of the United States Department of Agriculture requested that no action against Martin be taken until the Government's case against the B. & M. remedy had been tried as it was thought that the Government's case would be injured if Martin were deprived of his fellowship in the Massachusetts Medical Society and thereby membership in the American Medical Association on the plea that "The Medical Trust" was preventing the use by the public of a good therapeutic mixture.

Now that the trial is ended the committee have obtained the full stenographic notes of the testimony of the accused, in Baltimore—240 pages—and have sent him a "Form Letter", the first step in all proceedings against Fellows accused of breaking terms of the By-Laws or Code of Ethics, reciting the charges and asking for an explanation.

Faithfully yours,

WALTER L. BURRAGE,
Secretary of the Committee on Ethics and Discipline under the Terms of By-Laws.

THE SUSPENSION OF THE REGISTRATION OF DR. CALICCHIO AND OF DR. LACEY

The Commonwealth of Massachusetts
Department of
Civil Service and Registration
Board of Registration in Medicine
State House, Boston

August 27, 1932.

New England Journal of Medicine,

This is to inform you that at a hearing before the Board of Registration in Medicine, on Thursday, August 25, 1932, the license of Dr. Eli Calicchio, 21 Prince Street, Boston, Mass., was suspended for one month, for gross misconduct in the practice of his profession as shown by deceit in his testimony before the Industrial Accident Board.

The license of Dr. Henry O. Lacey, 402 Cambridge Street, Cambridge, Mass., was suspended for one month, for gross misconduct in the practice of his profession as shown by deceit in the matter of charging for services which he did not render.

Very truly yours,

STEPHEN RUSHMORE, M.D., *Secretary.*

IN MEMORIAM

EDWARD WYLLYS TAYLOR

A LAYMAN'S APPRECIATION

"The very finest human relationships are those between individuals; such relationships have from earliest times had their best exemplification in the attitude of the physician toward his patient. This at least is one of the heritages of the past which under no circumstances should be lost. There never has been a time when the opportunity of the practicing physician has been greater than now, and when there has been more need of the wise adviser in the purely confidential relation of man to man."

These words appeared some years ago in an edi-

torial of the *Boston Medical and Surgical Journal*, and were written by Dr. Edward Wyllys Taylor, who has recently died. Those who had the good fortune to have him for their personal physician and friend (we speak advisedly — for ill-health that brings one into relations of helpfulness and confidence with such a man as Dr. Taylor is not wholly *mis*fortune) know how completely the ideal therein expressed was exemplified by Dr. Taylor himself.

Today we are threatened by a perverted standard of values—with so much agitation of state medicine, free public clinics, and money-making private clinics. So that our chief dread of illness may well be lest it involves us in mechanized, institutionalized, or commercialized medical practice. Thus it is reassuring to recall that this tradition, expressed in words and realized in life-long practice by Dr. Taylor, is still cherished by those physicians who most truly represent that noblest of vocations whose primary purpose is to minister to human need.

E. C.

RECENT DEATH

TUPPER — Dr. Augustus Maclauchlan Tupper died at his home in Rockport, Massachusetts, August 22, 1932, aged 84. He had been spoken of as the dean of Cape Ann doctors and was widely known in that region.

He graduated from the Harvard University Medical School in 1870 and joined the Massachusetts Medical Society in 1875, retiring in 1930.

Dr. Tupper is survived by a son James Edgar Tupper and a daughter Mrs. George Grace both of Rockport.

NOTICE

A CORRECTION

In the article published in this *Journal*, August 18, 1932, under the title of "Routine Prophylactic Injection of Whole Blood in the New-Born. A Report of 800 Cases" by Roy J. Heffernan, M.D., the number on page 294, column two, fourteen lines from the bottom should read 800 instead of 200 as follows:

By January 10, 1932, the procedure had been used in 800 private cases.

NOTICE OF MEETING

NEW HAMPSHIRE SURGICAL CLUB

The thirty-fifth Annual Meeting of the New Hampshire Surgical Club will be held at the Farragut Hotel, Rye Beach, New Hampshire, on September 10 and 11, 1932.

This will be the first meeting of the club held on the seacoast since 1925 and should prove to be a welcome change to those members from the inland and northern parts of the state.

The scientific part of the meeting will take place on Saturday, September 10, to be followed by the banquet on the same evening. This is a decided change from the policy of previous years and it is to be hoped will prove successful.

A well-rounded program is underway that should prove attractive to all those attending.

A large attendance is expected and will be welcomed.

Wendell P. Clare, M.D., *Secretary*.

SOCIETY MEETINGS
CONGRESSES AND CONFERENCES

September 10 and 11—New Hampshire Surgical Club. See notice elsewhere on this page.

September 19-22 — International Congress of Biliary Lithiasis. See notice on page 427, issue of February 25.

September 26, 27, 28—Eighth Clinical Congress of the Connecticut State Medical Society. See page 331, issue of August 18.

September 30, October 1—Scientific Program for Annual Meeting of the New England Surgical Society. See page 333, issue of August 18.

October 12, 13 and 14—American Academy of Physical Therapy. See notice on page 384, issue of August 25.

October 17-21—Clinical Congress of the American College of Surgeons. See page 1330, issue of June 23.

October 17-28—The 1932 Graduate Fortnight of the New York Academy of Medicine. See page 1375, issue of June 30.

October 22-24—Health Education Institute. See notice on page 47, issue of July 7.

October 24-27 — American Public Health Association. See notice on page 1321, issue of December 31, 1931.

October 24-28—Inter-state Postgraduate Medical Association of North America. See page 39, issue of January 7.

BOOK REVIEW

The Lame, the Halt, and the Blind: The Vital Rôle of Medicine in the History of Civilization. By Howard W. Haggard, M.D. New York and London: Harper & Bros., 1932. xxiv + 420 pages.

This volume is similar in nature to a book already published by the same author, a few years ago, entitled "Devils, Drugs, and Doctors," one of the best selling non-fiction volumes of its time. Dr. Haggard's real message is to call to the attention of the general reading public that the history of the medical profession has been neglected by historians and that the effects of epidemics, changing forms of treatment, and new discoveries in medicine have made great and fundamental changes in civilization. The book is richly illustrated by unique pictures; written in a scientific spirit, although popular in style, it can be highly recommended.

BOOKS RECEIVED FOR REVIEW

Clio Medica edited by E. B. Krumbhaar. VIII. Physical Therapy by John S. Coulter. Published by Paul B. Hoeber, Inc. 142 Pages. Price $1.50.

Essentials of Pediatric Nursing by Ruth Alice Perkins. Second Edition. Published by F. A. Davis Co. 467 Pages.

Grundbegriffe der klinischen Medizin by Walter Marle. Published by Urban & Schwarzenberg. 640 Pages.

Everyday Practice Series edited by Harlow Brooks. *Functional Disturbances of the Heart* by Harlow Brooks. Published by J. B. Lippincott Co. 288 Pages. Price $5.00.